This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LIBRARY
OF
COOPER MEDICAL COLLEGE
DATE Dec 1905
NO. 73110 CLASS 20
GIFT OF
Dr Wm F. Cheney

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885
Dr. William F. Cheney

A TEXT-BOOK

OF THE

PRACTICE OF MEDICI

BY

DR. HERMANN EICHHORST

Professor of Special Pathology and Therapeutics and Director of the Medic
Clinic in the University of Zurich

AUTHORIZED TRANSLATION FROM THE GERMAN

EDITED BY

AUGUSTUS A. ESHNER, M.D.

Professor of Clinical Medicine in the Philadelphia Polyclinic ; Physician to th
Philadelphia Hospital ; Assistant Physician to the Orthopedic
Hospital and Infirmary for Nervous Diseases

With 85 Illustrations

LANE MEDICAL LIBRARY
7300
SAN FRANCISCO

VOLUME II

PHILADELPHIA AND LONDON
W. B. SAUNDERS & COMPANY
[illegible]

Copyright, 1901, by W. B. SAUNDERS & COMPANY.

Registered at Stationers' Hall, London, England.

ELECTROTYPED BY
THOMSON, PHILADA.

PRESS OF
W. B. SAUNDERS & COMPANY.

WTo
E34e
1901

CONTENTS OF VOLUME II.

PART V.—DISEASES OF THE NERVOUS SYSTEM (Continued).

PAGE

III. Diseases of the Medulla Oblongata . . . 17
- Preliminary Diagnostic Considerations . . . 17
- Chronic Progressive Bulbar Paralysis . . . 20
- Hemorrhage into the Medulla Oblongata . . . 25
- Embolism and Thrombosis of the Arteries of the Medulla Oblongata . . . 26
- Acute Inflammation of the Medulla Oblongata . . . 28
- Neoplasms of the Medulla Oblongata . . . 28

IV. Diseases of the Brain . . . 29
- Diagnostic Preliminary Considerations . . . 29
  - Focal Symptoms Attending Disease of the Cerebral Cortex . . 30
  - Focal Symptoms Attending Disease of the Centrum Semiovale . . . 34
  - Focal Symptoms Attending Disease of the Internal Capsule . 36
  - Focal Symptoms Attending Disease of the Cerebral Peduncles . 36
  - Focal Symptoms Attending Disease of the Pons . . . 37
  - Focal Symptoms Attending Disease at the Base of the Brain . 39
  - Symptom-complex Aphasia . . . 39
- Cerebral Anemia . . . 47
- Cerebral Hyperemia . . . 50
- Cerebral Edema . . . 52
- Cerebral Hemorrhage . . . 52
- Thrombosis and Embolism of the Cerebral Arteries . . . 60
- Inflammation of the Brain . . . 63
  - Cerebral Abscess . . . 63
  - Hemorrhagic Encephalitis . . . 66
  - Acute Cerebral Paralysis of Childhood . . . 67
  - Congenital Encephalitis . . . 68
- Cerebral Neoplasms . . . 68
- Cerebral Parasites . . . 72
- Aneurysms of the Cerebral Arteries . . . 73
- Hydrocephalus . . . 74
  - Congenital Hydrocephalus . . . 74
  - Acquired Hydrocephalus . . . 75
- Hypertrophy of the Brain . . . 77
- Atrophy of the Brain . . . 78
- Diffuse Sclerosis of the Brain . . . 78

Diseases of the Cerebral Meninges . . . 79
- Thrombosis and Inflammation of the Sinuses of the Brain . . . 79
- Internal Hemorrhagic Cerebral Pachymeningitis . . . 82
- Hemorrhage into the Cerebral Meninges . . . 84

V. Diseases of the Sympathetic Nerve . . . 85
- Paralysis of the Cervical Sympathetic . . . 85
- Irritation of the Cervical Sympathetic . . . 86

CONTENTS OF VOLUME II.

PAGE
entral Neuroses 86
ntral Neuroses with Motor Disturbances Predominating 87
Epilepsy 87
Tetany 94
Occupation-neuroses 98
Writers' Cramp 98
Chorea 100
Hereditary Chorea of Adults 105
Prehemiplegic Chorea and Posthemiplegic Chorea 105
Athetosis 106
Myoclonus 107
Paralysis Agitans 108
Tremor 112
Vertigo 112
Catalepsy 114
ntral Neuroses with Sensory Disturbances Predominating 115
Nervous Headache 115
Hemicrania 117
ntral Neuroses with Vasomotor and Trophic Disturbances Predominating 120
Vasomotor Neuroses of the Extremities 120
Intermittent Vasomotor Articular Neurosis 121
Intermittent Angioneurotic Edema 121
Symmetrical Gangrene 122
Perforating Ulcer 123
Progressive Facial Hemiatrophy 123
Facial Hemihypertrophy 125
Scleroderma 126
Myxedema 128
Exophthalmic Goiter 131
Akromegaly 136
ntral Neuroses with Psychic Alterations Predominating 139
Hysteria 139
Neurasthenia 144
Traumatic Neuroses 149

PART VI.—DISEASES OF THE MUSCLES.

Progressive Myopathic Muscular Atrophy 153
Pseudomuscular Hypertrophy 153
Juvenile Muscular Atrophy 155
Infantile Muscular Atrophy 157
Neural Progressive Muscular Atrophy 158
True Muscular Hypertrophy 159
Progressive Ossifying Myositis 160
Polymyositis 161
Myotonia 162
Myasthenia 163

PART VII.—DISEASES OF THE SKIN.

nflammations of the Skin 164
ythematous Inflammations of the Skin 164
Urticaria 164

PAG
- Vesicular Inflammations of the Skin 17
  - Eczema 17
  - Miliaria 17
  - Cheiropompholyx 17
- Bullous Inflammations of the Skin 17
  - Pemphigus 17
  - Hereditary Bullous Epidermolysis 17
  - Exfoliative Dermatitis 17
- Pustular Inflammations of the Skin 17
  - Impetigo 17
  - Acne Vulgaris 18
  - Acne Mentagra 18
  - Acne Rosacea 18
- Scaly Inflammations of the Skin 18
  - Psoriasis 18
  - Pityriasis Rubra 18
- Papular Inflammations of the Skin 19
  - Prurigo 19
  - Lichen of the Scrofulous 19
  - Lichen Ruber 19

II. Secretory Disorders of the Skin 19
- Secretory Disorders of the Sweat-glands 19
  - Increased Secretion of Sweat 19
  - Diminished Secretion of Sweat 19
  - Alterations in the Character of the Sweat 19
- Secretory Disorders of the Sebaceous Glands 19
  - Increased Sebaceous Secretion 19
  - Diminution in the Sebaceous Secretion 19
  - Disorders of the Sebaceous Secretion 19
    - Comedo 19
    - Cutaneous Sand 19

III. Hypertrophies of the Skin 19
- Hypertrophies of the Pigment of the Skin 19
  - Nevus 19
  - Chloasma 19
- Hypertrophy of the Epidermis 20
  - Ichthyosis 20
  - Acanthosis Nigricans 20
- Hypertrophy of the Hair 20
- Hypertrophy of the Nails 20

IV. Atrophy of the Skin 20
- Atrophy of the Pigment of the Skin 20
- Atrophic Changes in the Hairs 20
  - Graying of the Hair 20
  - Deficiency of Hair 20
  - Circumscribed Alopecia 20
  - Brittleness of the Hair 20
- Atrophic Disorders of the Nails 20
- Atrophy of the Cutis 20

V. Cutaneous Neuroses 20
  - Itching of the Skin 20

VI. Parasites of the Skin 211
- Animal Parasites of the Skin 211
  - Itch 211
  - Acarus Folliculorum 21
  - Pediculi 21
    - Head-louse 21
    - Crab-louse 21
    - Clothing-louse 21

PAGE
Fleas 220
Bedbug 220
Cysticercus of the Subcutaneous Connective Tissue 220
Vegetable Parasites of the Skin 221
Pityriasis Versicolor 221
Favus 222
Herpes Tonsurans 226
Erythrasma 229

PART VIII.—DISEASES OF THE SPLEEN AND THE BLOOD.

I. Diseases of the Spleen 230
Embolic Infarction of the Spleen 230
Abscess of the Spleen 231
Perisplenitis 233
Amyloid Spleen 234
Carcinoma of the Spleen 235
Echinococcus of the Spleen 235
Rupture of the Spleen 236
Wandering Spleen 236
Aneurysm of the Splenic Artery 238
II. Diseases of the Blood 238
Leukemia 238
Pseudoleukemia 247
Progressive Pernicious Anemia 250
Chlorosis 258
Purpura 262
Scorbutus 266
Bleeders' Disease 270
Paroxysmal Hemoglobinuria 272

PART IX.—DISORDERS OF METABOLISM.

Obesity 275
Gout 279
Diabetes Mellitus 285
Diabetes Insipidus 296
Rachitis 299
Osteomalacia 305
Deforming Arthritis 308

PART X.—INFECTIOUS DISEASES.

INFECTIOUS DISEASES OF TYPICAL LOCALIZATION.

I. Acute Infectious Exanthemata 311
Typhus Fever 311
Measles 316
Rötheln 322
Scarlet Fever 323
Erysipelas 329
Herpes Zoster 334
Miliary Fever 338
Varicella 338
Variola 341
II. Infectious Diseases with Local Alterations in the Locomotor Apparatus 349
Acute Articular Rheumatism 349
Chronic Articular Rheumatism 354
Muscular Rheumatism 356

PAGE

III. Infectious Diseases with Local Alterations in the Blood and Blood-generating Organs . . . 357
- Relapsing Fever . . . 357
- Malarial Fever . . . 361
- Bubonic Plague . . . 371

IV. Infectious Diseases with Local Alterations in the Respiratory Organs . . . 374
- Whooping-cough . . . 374
- Influenza . . . 380

V. Infectious Diseases with Local Alterations in the Digestive Organs . . . 384
- Epidemic Parotitis . . . 384
- Acute Phlegmonous Pharyngitis . . . 387
- Typhoid Fever . . . 387
- Dysentery . . . 403
- Asiatic Cholera . . . 409
- European Cholera . . . 420

VI. Infectious Diseases with Local Alterations in the Sexual Organs . . . 421
- Gonorrhea . . . 421
- Soft Chancre . . . 436

VII. Infectious Diseases with Local Alterations in the Nervous System . . . 442
- Epidemic Cerebrospinal Meningitis . . . 442
- Secondary Purulent Cerebrospinal Meningitis . . . 448
- Serous Cerebrospinal Meningitis . . . 449
- Tetanus . . . 449

INFECTIOUS DISEASES OF VARYING LOCALIZATION.

I. Diphtheria . . . 456
- Pharyngeal Diphtheria . . . 456
- Laryngeal Diphtheria . . . 464

II. Tuberculosis . . . 470
- Chronic Pulmonary Tuberculosis . . . 470
- Chronic Laryngeal Tuberculosis . . . 486
- Chronic Pharyngeal Tuberculosis . . . 488
- Chronic Intestinal Tuberculosis . . . 488
- Chronic Tuberculosis of the Kidneys and the Bladder . . . 491
- General Miliary Tuberculosis . . . 495
- Tuberculous Cerebrospinal Meningitis . . . 498
- Tuberculous Peritonitis . . . 502
- Tuberculous Pleuritis and Pericarditis . . . 505
- Scrofulosis . . . 506
- Solitary Tuberculosis . . . 510

III. Syphilis . . . 510
- Acquired Syphilis . . . 510
  - Acquired Syphilis in the Primary and the Secondary Stage . 510
  - Tertiary Syphilis . . . 522
    - Tertiary Syphilis of the Skin, the Muscles, the Fasciæ, the Bursæ, the Bones, and the Joints . . . 522
    - Tertiary Syphilis of the Respiratory Organs . . . 524
    - Tertiary Syphilis of the Digestive Organs . . . 526
    - Tertiary Syphilis of the Genito-urinary Organs . . . 528
    - Tertiary Syphilis of the Circulatory Organs . . . 528
    - Tertiary Syphilis of the Nervous System . . . 529
- Hereditary Syphilis . . . 532

IV. Leprosy . . . .

V. Zoonoses . . . .
  Anthrax . . . .
  Glanders . . . .
  Actinomycosis . . . .
  Foot-and-mouth Disease . . . .
  Rabies . . . .

INDEX TO VOLUME II. . . . .
GENERAL INDEX . . . .

DISEASES OF THE NERVOUS SYSTEM

(CONTINUED).

III. DISEASES OF THE MEDULLA OBLONGATA.

PRELIMINARY DIAGNOSTIC CONSIDERATIONS.

THE medulla oblongata is known also as the *spinal bulb*, and its diseases and also its distinctive symptoms are therefore designated *bulbar*. As the motor and sensory tracts between the brain and the spinal cord traverse the medulla oblongata, it is not surprising that in the presence of bulbar disease *motor and sensory disturbances* are not rarely encountered. It is worthy of especial consideration that the motor tracts from the brain largely cross over within the *pyramidal decussation* to the lateral pyramidal tract of the opposite half of the spinal cord, for it follows that if the motor tract is interrupted before decussation has taken place the paralysis will occur upon the opposite side of the body, whereas lesions below this point (toward the spinal cord), therefore after decussation has taken place, give rise to paralysis upon the same side of the body. At times lesions are so situated as to involve a portion of the motor path above and another portion below the decussation, and as a result of which it is possible that perhaps the arm upon the side of the lesion (below the decussation) and the leg upon the opposite side of the body (above the decussation) are paralyzed—so-called *crossed hemiplegia*. Smaller lesions give rise at times to *bulbar monoplegia* of the arm or the leg, and it may also happen that the paralysis occurs upon the same side as the lesion or upon the opposite side accordingly as the lesion is seated above or below the decussation of the motor paths.

Motor and sensory disturbances are in nowise characteristic of bulbar disease. The *bulbar symptoms* proper result from involvement of the *nuclei* of individual *cerebral nerves*, particularly the hypoglossal, the glossopharyngeal, and the vago-accessory, which are situated at the lowermost extremity of the fourth ventricle. As is well known, groups of ganglion-cells are found upon the floor of the fourth ventricle, from which arise nerve-fibers for the individual cerebral nerves, whence the designation nuclei of cerebral nerves. These nerve-fibers first pass through the medulla oblongata (and the pons) before they appear at the surface as cerebral nerve-roots, and they thus pursue an intramedullary (bulbar, pontine) course. Between the nuclei and the brain the nerve-fibers undergo decussation, so that, for example, the fibers of the right facial nerve above the facial nucleus pass over to the tracts from the left cerebral hemisphere. From these relations it will be clear that lesions in the nerve-nuclei or within the medullary path up to the point of exit of the cerebral nerve-trunk will give rise to paralysis upon the same side, while disease above the nuclei for the cerebral nerves will be attended with paralysis upon the opposite side of the body.

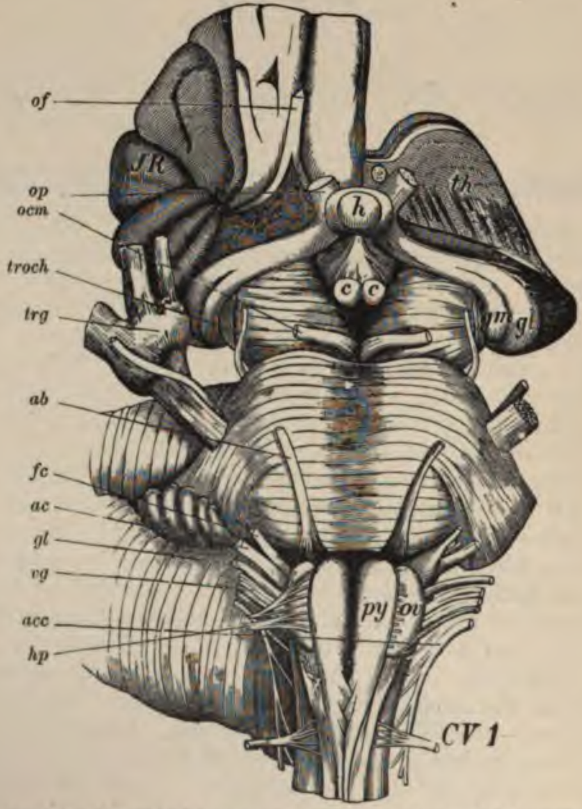


FIG. 1.—Medulla oblongata and adjacent portion of the brain, with the nerves arising therefrom (after Schwalbe): *of*, olfactory nerve; *op*, optic nerve; *ocm*, oculomotor nerve; *troch*, trochlear nerve; *trg*, trigeminal nerve; *ab*, abducens nerve; *fc*, facial nerve; *ac*, auditory nerve; *gl*, glossopharyngeal nerve; *vg*, vagus; *acc*, accessory nerve; *hp*, hypoglossal nerve; *py*, pyramid; *ov*, olive; *th*, optic thalamus; *gm*, *gl*, internal and external geniculate bodies; *h*, hypophysis; *c*, *c*, mammillary bodies; *JR*, island of Reil.

The principal bulbar symptoms consist in *derangement of* *articulation*, *paralysis of swallowing*, and *disturbance in breathing* and in *cardiac action*. *Articulatory disturbances* (*anarthria*, *dysarthria*) result from paralysis of the hypoglossal nerve and the attendant paralysis of the tongue (bulbar glossoplegia). In severe cases the tongue lies in the mouth like an immovable mass of flesh, and the formation of a bolus is therefore impossible. Should the disease pursue a chronic course, degenerative atrophy of the tongue takes place, and the organ exhibits fibrillary muscular contractions and degenerative electric reaction. The symptoms are entirely analogous to those of anterior poliomyelitis, as the ganglion-cells of the nuclei of the cerebral nerves also are largely endowed with trophic-motor functions. *Disorders in swallowing* depend upon paralytic conditions involving the glossopharyngeal and vago-accessory nerves, which supply the muscles of deglutition with motor fibers. Vago-accessory paralysis may be attended also with *paralysis of the laryngeal muscles*, *derangement in respiratory activity* (Cheyne-Stokes breathing), and *acceleration of the action of the heart*. Naturally, not all bulbar diseases give rise to the bulbar symptoms just described. These occur only when the bulbar nuclei of the cerebral nerves or the intrabulbar nerve-fibers originating from them are involved in the morbid process.

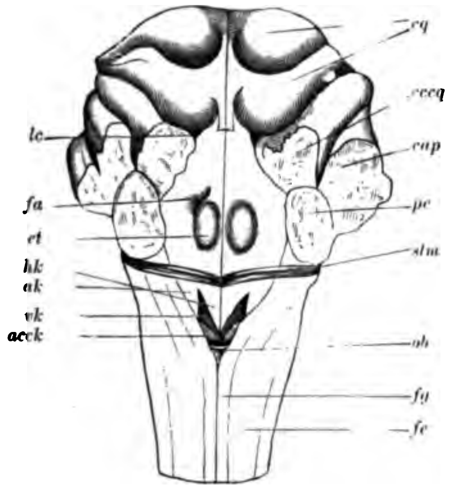


FIG. 2.—Medulla oblongata seen from its posterior aspect, with a view of the fourth ventricle and the nerve-nuclei upon its floor: *lc*, locus cœruleus (nucleus of the trigeminus); *fa*, fovea anterior (nucleus of the facial nerve); *et*, eminentia teres (knee of the facial nerve); *hk*, nucleus of the hypoglossal nerve; *ak*, median nucleus of the posterior root of the auditory nerve; *vk*, nucleus of the vagus (ala cinerea); *acck*, nucleus of the accessory nerve; *cq*, corpora quadrigemina; *cccq*, crura cerebelli ad corpora quadrigemina; *cap* crura cerebelli ad pontem; *pc*, pedunculus cerebelli; *stm*, medullary or auditory striæ *ob*, obex; *fg*, funiculus gracilis; *fc*, funiculus cuneiformis.

For the better comprehension of what has preceded, a few *anatomic remarks and illustrations* may be appropriate. Upon the anterior (ventral) aspect the limits of the medulla oblongata can be accurately defined. The medulla begins below in the pyramidal decussation and terminates above at the anterior (inferior) border of the pons (Fig. 1). Upon the posterior (dorsal, ventricular) aspect there is no sharp boundary between the medulla oblongata and the pons. From the purely topographic point of view this boundary would correspond about with the situation of the medullary or auditory striæ (p. 19, Fig. 2, au). A sharp division between the medulla oblongata upon the one hand, and the spinal cord and the pons, upon the other hand, is of but little significance upon the ventricular aspect, as the nuclei of some of the nerves are continued from the lower portions of the medulla deeply into the spinal cord (accessory nucleus), while at the upper boundary some nerve nuclei belong both to the medulla and to the pons. The *hypoglossal nucleus* is situated in the lowermost portion of the fourth ventricle, immediately adjacent to the median fissure (p. 19, Fig. 2, *hk*), and in close proximity are the nuclei for the vagus and the accessory (Fig. 2, *vk*, *acck*). The *glossopharyngeal nucleus* is more deeply situated in the medulla, just above the nucleus for the vagus. The facial nucleus is separated by a considerable distance from the nuclei of the cerebral nerves previously mentioned, and is situated above the medullary striæ in the depth of the [illegible] (Fig. 2, [illegible]). Fig. 3, p. 21, represents a transverse section through the medulla oblongata at about the level of the middle of [illegible] the floor of the fourth ventricle in this situation may [illegible] the nuclei of the hypoglossus and the vagus, together with [illegible] fibers.

CHRONIC PROGRESSIVE BULBAR PARALYSIS (PROGRESSIVE INFERIOR POLIO-ENCEPHALITIS).

Anatomic Alterations.—The [illegible] alterations attend- [illegible] wholly analogous [illegible] atrophy. The condi- [illegible] degeneration [illegible] disappearance of the motor trophic ganglion-cells [illegible] the cerebral [illegible] (glosso-pharyngeal) and [illegible] is mani- [illegible] nerve- [illegible] grayish [illegible] peripheral [illegible] the [illegible] bands, [illegible] many [illegible] have disappeared [illegible] remains [illegible]

[illegible] are studied [illegible] tion, the ganglion-cells [illegible] with yellowish [illegible] brownish granular pigment, and [illegible] plump structures, in part without processes. [illegible] progressive diminution in size. Finally they disappear entirely and are replaced by neuroglia. Microscopic examination of the medulla oblongata is indispensable, as the tissues appear unaltered on macroscopic examination. Further, degeneration in the pyramidal tracts is often [illegible] observed.

Etiology.—Experience has shown that chronic progressive bulbar paralysis is *more common in men than in women*, and that it develops generally *between the fortieth and the sixtieth year of life.* Occasionally it occurs in the course of **infectious diseases** (syphilis, influenza). Some patients attribute their disorder to **exposure to cold, traumatism, emotional disturbances,** and **over-exertion on the part of the muscles of the lips,** as, for instance, in blowing. At times the disease develops in connection with **spinal progressive muscular atrophy** or with **amyotrophic lateral sclerosis.**

Symptoms.—Progressive chronic bulbar paralysis almost always develops slowly, and pain and a sense of contraction in the neck and the occiput, as well as vertigo and headache, often occur as **prodromes.** The first paralytic symptoms generally appear in the tongue, then changes in the lips are added, and, finally, the muscles of the palate, the pharynx, and the larynx are involved.

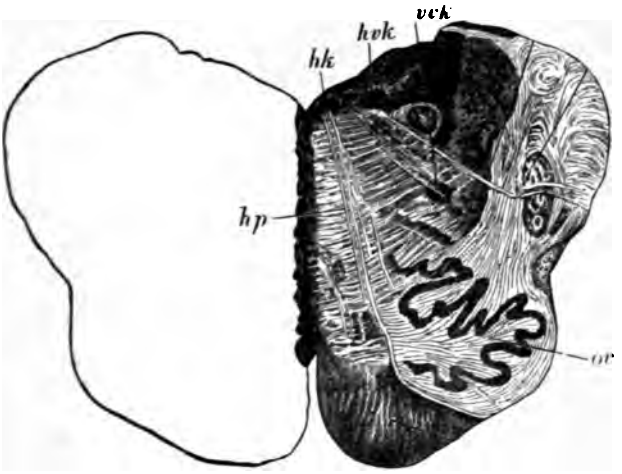


FIG. 3.—Transverse section through the medulla oblongata at about the level of the middle of the olive: enlarged four times: *hk*, hypoglossal nucleus; *hp*, course for the fibers of the hypoglossal nerve; *hvk*, posterior nucleus of the vagus; *vvk*, anterior nucleus of the vagus; *vg*, course for the fibers of the vagus; *atrv*, ascending root of the trigeminus; *ov*, olive; *py*, pyramid.

Only rarely are the *nuclei of other nerves*, as, for instance, the *nucleus of the abducens*, which lies close to the facial nucleus, or the motor *nucleus of the trigeminus*, which lies below the locus cœruleus (p. 19, Fig. 2, *lc*), involved. In the latter event paralysis of the muscles of mastication would result.

The patients generally are conscious earliest of **impaired motility and undue readiness of fatigue of the tongue** in speaking and in chewing. These difficulties increase, and soon become appreciable as **articulatory disturbances,** involving especially the linguals (r, sch, s, **l, k, g, d, t, w, j**). At the same time there is great difficulty in

forming the bolus of food. The patient is unable to apprehend with the tip of the tongue food that has become lodged between the cheeks and the teeth, but he is compelled to push it forward with the fingers; and, besides, the tongue has lost its property of forcing the bolus into the pharynx, so that the food must be pushed backward with the fingers or with a spoon or a wooden rod. If the patient be requested to protrude the tongue or to move it from side to side, this can be done but imperfectly and with difficulty, if at all. The ability and the power to retract the tongue are preserved for a relatively long time. The tongue is conspicuously atrophied, and it exhibits more or less marked fascicular (fibrillary) muscular contractions. As a result of the atrophy of the muscular structure of the tongue its surface presents a wrinkled appearance.

Shortly after involvement of the muscles of the tongue alterations take place in the **orbicular muscle of the mouth,** whose nerve-fibers are believed by some physicians to be derived, not from the facial, but from the hypoglossal, nucleus. The **lips** appear unusually thin, and this may be readily recognized by grasping them between the fingers. At the same time the **buccal fissure** remains open and appears enlarged laterally. The patient is unable to pucker his lips, and cannot therefore whistle. The **articulation of labials** (b, p, m, w, o, u) is impaired or impossible. Frequently **saliva** can be seen to dribble from the open mouth, and the more so as the swallowing of this fluid is interfered with. As the medulla oblongata contains nerve-centers controlling the secretion of saliva, increased discharge may be associated with increased secretion of saliva. The **change in the facial expression** is most striking. The frontal muscles do not participate in the atrophy and paralysis, and the forehead is thrown into deep horizontal folds. The muscles of the cheek and the chin, however, are wasted and feeble, and the nasolabial folds are well developed. While the frontal region presents an expression of surprise, the lower half of the face appears sad and painfully distorted (Fig. 4).

Paralysis of the muscles of the soft palate frequently gives rise to difficulty in swallowing, because in consequence of imperfect closure of the nasopharyngeal cavity the ingested material passes readily from behind into the nose. The formation of the letters b and p is also interfered with if the current of air can escape through the nose without obstruction, and the letters sound like me, we, or fe. These are given their natural sound, however, if the nasal orifices are closed with the fingers during the act of speaking. On examination the arch of the palate will be seen to hang downward in a flaccid manner, and it moves to and fro like a curtain when respiration is active. The difficulty in swallowing becomes materially aggravated when the *depressor of the epiglottis* is involved in the paralysis, so that, in swallowing, the entrance into the larynx remains open and food can readily find its way into the cavity of the larynx. The condition is attended with the danger that food laden with bacteria may gain entrance into the smaller bronchi and pulmonary alveoli, and give rise to inspiration-pneumonia and pulmonary abscess and gangrene. Also, *paralysis of the muscles of the esophagus* interferes in marked degree with the act of deglutition. If the patient is not careful in eating, the esophagus may be excessively distended with food, exert pressure upon the larynx and the vago-accessory nerve, and give rise to alarming dyspnea and palpitation of the heart. *Paralysis of the muscles of the vocal bands* interferes with phonation; the voice becomes feeble, masked, monotonous, and high-pitched. Should bilateral paralysis of the recurrent nerve be present, the patients may only be able to talk unintelligibly, and to communicate with those about them solely by writing.

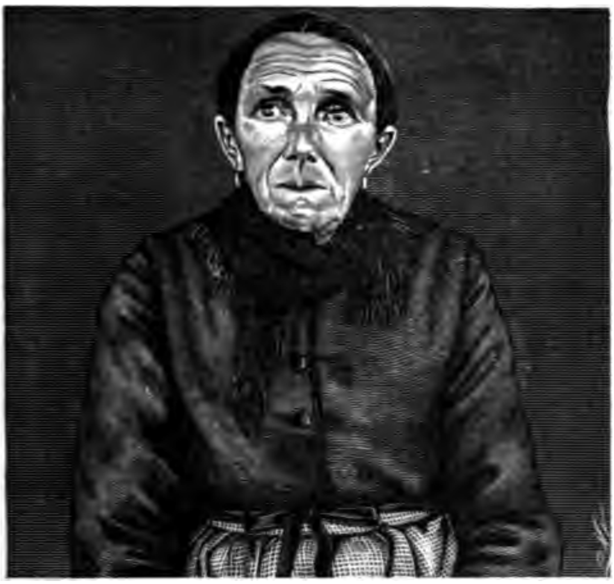


FIG. 4.—Facial expression in a case of chronic progressive bulbar paralysis in a woman 45 years old; from a photograph (personal observation, Zurich clinic).

At times changes in the action of the heart can be demonstrated, at first frequently slowing of the pulse (irritation of the vagus), subsequently acceleration of the pulse (paralysis of the vagus). Attacks of intense dyspnea also occur, and at times lead to a fatal termination. In all of the atrophic and paralyzed muscles accessible to examination degenerative electric reaction, exaggeration of mechanical irritability, and loss of reflex movement will be found. Cutaneous sensibility remains unchanged. The mental state likewise exhibits no material disturbance, except that frequently boisterous and irrepressible laughter and uncontrollable weeping occur frequently.

The *duration of the disease* generally extends over from one to three years. Death results from excessive marasmus or from inspiration-pneumonia and its consequences, or from paralysis of the heart or from intercurrent coma. At times the morbid process extends to the motor-trophic ganglion-cells in the anterior horns of the spinal cord, so that spinal progressive muscular atrophy becomes superadded to chronic bulbar paralysis. Weakness of the muscles of the neck develops with especial constancy, so that the head falls forward and must be supported by the hands of the patient.

Diagnosis.—The recognition of chronic bulbar paralysis is easy in view of its slow and regular development.

The disorder is distinguished from *hemorrhage*, *embolism*, and *thrombosi* in the medulla oblongata by the sudden onset and generally fatal termina tion of these latter. *Neoplasms* and *aneurysms*, as a rule, cause unilateral o predominantly unilateral paralytic symptoms affecting bulbar nerves, ar in addition, vomiting and choked disc are frequently present. At ti bilateral lesions in the cerebrum, and principally in the lenticular nucl or in the frontal convolutions, give rise to symptoms of bulbar paralys so-called *pseudobulbar paralysis* or *cerebral bulbar paralysis*. The sympt of this disorder, however, set in with apoplectiform phenomena, whil paralysis does not occur simultaneously on both sides, but successively extremities also are involved, and the paralyzed muscles do not ex degenerative atrophy and degenerative electric reaction. Under the nation *myasthenic bulbar paralysis* bulbar symptoms have been describ result from abnormal muscular fatigue. The patient is able to exec given movements at first, but tires readily, and then muscular p occurs, until the muscles have recuperated. Generally the muscles o the eyes, the trunk, and the extremities are involved. The condition is a disease of the muscles. Occasionally *congenital bulbar paralysis* is observed, which improves spontaneously or as the result of electric treatment.

Prognosis.—Chronic bulbar paralysis is incurable, and death appears unavoidable.

Treatment.—**Causal treatment** should be employed in cases in which *syphilis* has been present (mercurial inunctions, potassium iodid internally). All possible *nervines*, *electricity*, and *baths* have been employed, but all without permanent success. Marked difficulty in deglutition may require **feeding through the stomach-tube**, and threatening suffocation **intubation** or **tracheotomy**.

The designation *superior polio-encephalitis* has been applied to cases in which the nuclei of the nerves for the ocular muscles are diseased, and of which, as is well known, the nucleus of the abducens is situated in the medulla oblongata in the vicinity of the facial nucleus, while the trochlear and the oculomotor nuclei are situated in the aqueduct of Sylvius. Disease of these nuclei will give rise to paralysis of the external ocular muscles, although the elevator of the upper lid is frequently involved in slight degree. The internal ocular muscles (sphincter of the iris, tensor of the choroid), however, usually remain unaffected, and accordingly the reflex and the accommodative activity of the pupils remains unaltered. The disease has therefore been designated also *external nuclear ophthalmoplegia.* It may develop in an *acute, subacute,* or *chronic manner*, and, particularly in the last-named event, one ocular muscle after another may be attacked, so that the condition has been spoken of as *chronic progressive ophthalmoplegia.*

Acute ophthalmoplegia frequently sets in with headache, vertigo, and stupor, and death results with progressive loss of consciousness. Improvement, and even recovery, may, however, occur. At times the morbid process extends to the bulbar nuclei, so that the symptoms of progressive bulbar paralysis are superadded to those of external progressive ophthalmoplegia.

In the way of *anatomic alterations* hemorrhage into the nuclei of the nerves for the ocular muscles and degeneration of the ganglion-cells have been observed in acute and subacute cases, and degeneration of the ganglion-cells alone in chronic cases, although observations are on record according to which no anatomic alteration could be discovered, so that the symptoms of superior polio-encephalitis at times appear to be due to a neurosis.

Among the *causes, toxic influences* may be mentioned (alcoholism, intoxi-ation with lead, carbon monoxid, sulphuric acid, sausage-poisoning, dia-etes mellitus). The disorder has at times occurred in the sequence of *fectious diseases* (pneumonia, articular rheumatism, influenza). *Exposure cold, traumatism,* and *fright* have also been mentioned as causes. The isease has not rarely developed in the course of other *nervous diseases*, as, instance, tabes dorsalis, multiple cerebrospinal sclerosis, progressive al muscular atrophy, chronic bulbar paralysis, exophthalmic goiter, progressive paralysis of the insane.

Treatment with potassium iodid is said to be useful. For details, refer- should be made to text-books of ophthalmology.

ORRHAGE INTO THE MEDULLA OBLONGATA.

iology, Symptoms, and Diagnosis.—Hemorrhage into dulla oblongata is uncommon, and probably results from rupture of so-called **miliary aneurysms.** In accordance with the size of the hemorrhage a distinction is made between *capillary* and *focal hemorrhage.* Focal hemorrhage of considerable extent causes speedy or sudden death through paralysis of the center for the vagus. Smaller focal hemorrhage gives rise to the most varied symptoms, accordingly as the motor or sensory tracts or the bulbar nuclei of the cerebral nerves are destroyed. In diagnosis the sudden occurrence of paralytic symptoms referable to the bulbar nuclei—so-called *acute* or *apoplectic bulbar paralysis*—is characteristic. If the pyramidal tract is involved in addition to the nuclei, it may happen that the paralysis of the cerebral nerves is upon the same side as the lesion and that of the extremities upon the opposite side of the body—so-called *alternating hemiplegia.*

The essential facts with regard to the possible peculiarities of motor paralysis have already been stated in the diagnostic preliminary considerations. Further, paralysis of all four extremities may occur as the result of bulbar hemorrhage, as the motor pyramidal tracts lie close together in the medulla oblongata.

The **prognosis** is unfavorable. Difficulty in deglutition may readily give rise to inspiration-pneumonia and its consequences, and, in addition, there may be danger of paralysis of the vagus.

The **treatment** consists in the application of **ice-bags to** both sides of the occiput, and feeding by **enemata,** and subsequently through the **stomach-tube.**

EMBOLISM AND THROMBOSIS OF THE ARTERIES OF THE MEDULLA OBLONGATA.

Etiology.—Occlusion of the arteries of the medulla oblongata with plugs conveyed by the blood-stream—*emboli*—or by masses of fibrin that have formed *in situ*—*thrombi*—gives rise to necrotic softening of the tissue deprived of its blood-supply, and which, in accordance with its age, exhibits a varied color, and is designated *red, yellow,* and *gray* softening. The condition is not rarely one of multiple foci of softening, which may possibly be present also in the pons and the cerebrum. Smaller foci may undergo absorption, with the formation of *cicatrices,* while larger give rise to *cysts,* which are usually filled with clear fluid. **Acute endocarditis** or **valvular disease of the heart** generally constitute the source for emboli; less commonly, thrombotic deposits upon the intima of the aorta in the presence of *arteriosclerosis* or *aortic aneurysm.* Thrombosis, however, occurs as a result of arteriosclerosis of the medullary arteries, and less commonly as a result of arterial compression. **Syphilis** is not a rare cause for arteriosclerosis, especially for that involving the basilar artery.

Symptoms and Diagnosis.—Embolism and thrombosis of the medullary arteries may cause *sudden death* if they give rise to rapid paralysis of the nucleus of the vagus. In other instances the clinical picture of **acute** or **apoplectiform bulbar paralysis** develops, and then the difficult diagnostic problem arises of determining whether this is due to embolism or thrombosis, and not to hemorrhage or acute inflammation. This problem can never be solved with certainty. Although the detection of valvular disease of the heart is rather indicative of embolism, and that of antecedent syphilis, particularly in young persons, points to thrombosis, hemorrhage may take place under either condition.

The symptoms of bulbar embolism and thrombosis vary in accordance with the vascular area obstructed, and a correct opinion is possible only from a knowledge of the *blood-supply.* The medulla oblongata receives its vessels from the two *vertebral arteries* and from the *basilar artery* resulting from their union. The trunk of the vertebral arteries gives branches only to the bulbar nerve-trunks, while from it originates the *anterior spinal artery,* which surrounds the nuclei of the hypoglossal and accessory nerves with a capillary network (Fig. 5). In addition, each vertebral artery gives off a *posterior inferior cerebellar artery,* which supplies the olive and the choroid plexus of the fourth ventricle, as well as the pyramidal tract, with blood, either directly or through the intermediation of the *posterior spinal artery* arising from it. Finally, the *basilar artery*, through branches from its lower portion, supplies the nuclei of the vagus, the glossopharyngeal, and the auditory nerve with blood, while from its upper portion branches are given off for the nuclei of the facial, abducens, oculomotor, and trochlear nerves. Obstruction of the lower portion of the basilar artery is particularly dangerous, on account of the likelihood of paralysis of the vagus. Bulbar paralysis will be most extensive when a thrombus obstructs the basilar and the vertebral arteries simultaneously. According to Griesinger, this occurrence, as well as obstruction of the basilar artery alone, can be recognized from the fact that pressure upon both carotids

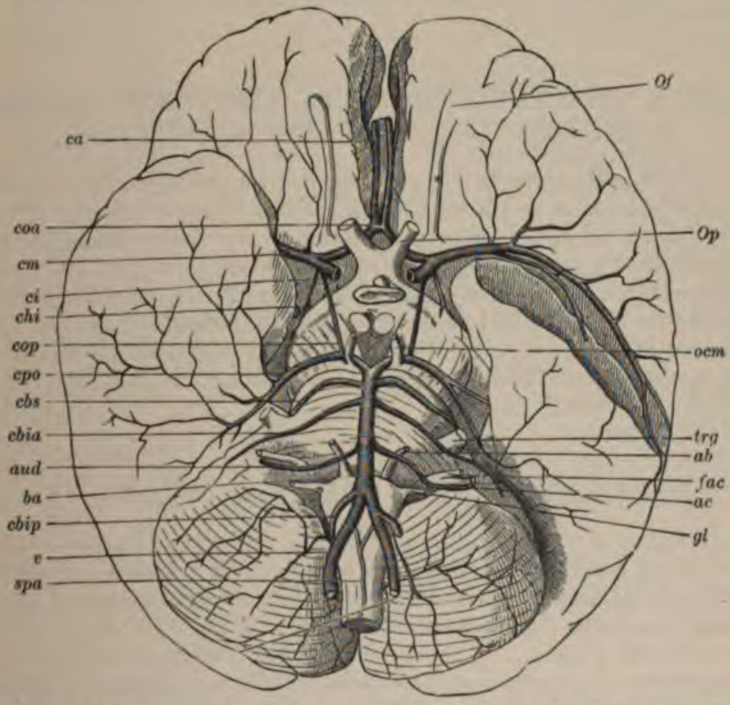


FIG. 5.—Distribution of the arteries at the base of the brain (after Henle): *ca*, anterior cerebral artery or artery of the corpus callosum; *coa*, anterior communicating artery; *cm*, middle cerebral artery or artery of the fissure of Sylvius; *ci*, internal carotid artery; *chi*, choroid artery; *cop*, posterior communicating artery; *cpo*, posterior cerebral artery; *cbs*, superior cerebellar artery; *cbia*, anterior inferior cerebellar artery; *aud*, auditory artery; *ba*, basilar artery; *cbip*, posterior inferior cerebellar artery; *v*, right vertebral artery; *spa*, anterior spinal artery; *spp*, posterior spinal artery; *Of*, olfactory nerve; *Op*, optic nerve; *ocm*, oculomotor nerve; *trg*, trigeminal nerve; *ab*, abducens nerve; *fac*, facial nerve; *ac*, auditory nerve; *gl*, glossopharyngeal nerve.

may be attended with loss of consciousness and general clonic muscular spasm, in consequence of cerebral anemia, although these phenomena at times appear also in persons with a patulous basilar artery.

The **treatment** is the same as that for hemorrhage (p. 26). Antecedent syphilis will require inunctions of mercurial ointment (5.0—75 grains—daily) and the administration of potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily).

ACUTE INFLAMMATION OF THE MEDULLA OBLONGATA (ACUTE BULBAR MYELITIS).

Acute bulbar myelitis is extremely rare, and occurs principally in the form of multiple small hemorrhagic foci, which in part are discovered only on microscopic examination. Fatty granule-cells, degenerated and disintegrating nerve-fibers, thickening and fatty degeneration of the vessels, and emigrated red blood-corpuscles constitute the principal elements of the inflammatory focus. The disease is attended with slight fever, and pursues the course of an acute or apoplectiform bulbar paralysis, and hitherto always with a fatal termination.

NEOPLASMS OF THE MEDULLA OBLONGATA.

Neoplasms occur but rarely in the medulla oblongata. One or several *tuberculous nodules* are present with relative frequency, although gliomata, fibromata, myxomata, papillomata, and gummata also have been observed. The new-growths may acquire the size of a walnut. At times they are wholly *unattended with symptoms*. In other instances *general* (*diffuse*) *cerebral symptoms* appear, particularly headache, vertigo, vomiting, eructation, choked disc, clonic convulsions, stupor, and progressive loss of memory. The gradual development of *bulbar symptoms* is alone distinctive of bulbar neoplasms, and these symptoms are generally due to increasing pressure upon the individual bulbar nuclei or upon the nerve-roots of the individual cerebral nerves arising from them. There is usually present paralysis of the tongue, the palate, the esophagus, and the laryngeal muscles.

The **diagnosis** is difficult. It is impossible to determine whether a neoplasm is situated in the medulla oblongata itself or has invaded the bulb from the vicinity.

The **prognosis** is unfavorable, as the **treatment** is promising of success only from the use of antisyphilitic measures in the presence of gummata.

IV. DISEASES OF THE BRAIN.

DIAGNOSTIC PRELIMINARY CONSIDERATIONS.

For the satisfactory diagnosis of diseases of the brain answers must be given to three questions, namely: 1. Is disease of the brain at all present? and, if so, 2. What is its seat? and, finally, 3. Upon what anatomic alterations does it depend?

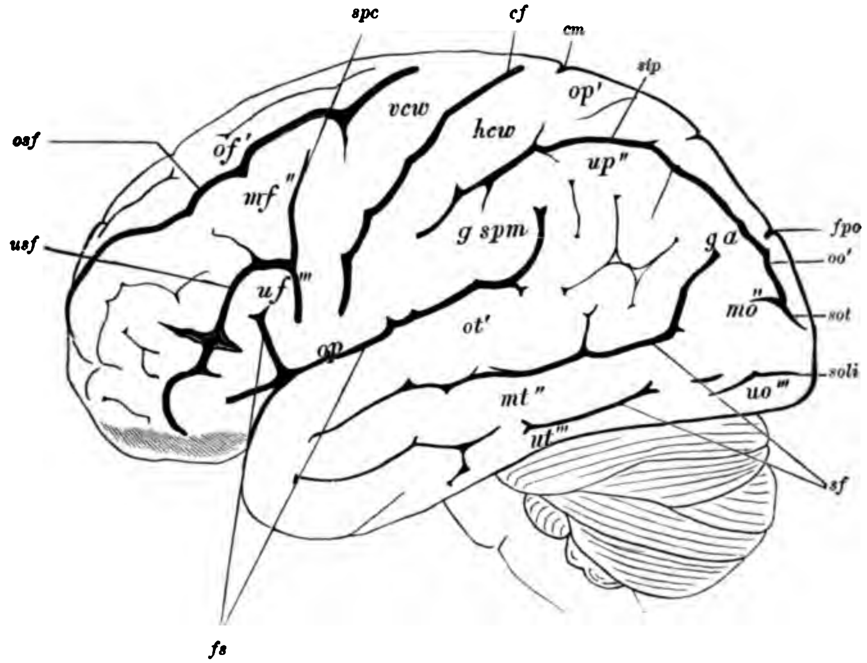


Fig. 6.—Convolutions of the convexity of the brain (after Ecker): *of'*, superior (or first) frontal convolution; *mf''*, middle (or second) frontal convolution; *uf'''*, inferior (or third) frontal convolution; *vcw*, anterior central (or ascending frontal) convolution; *hcw*, posterior central (or ascending parietal) convolution; *op'*, superior parietal lobule; *up''*, inferior parietal lobule, of which *gspm* represents the supramarginal and *ga* the angular gyrus; *ot'*, superior (or first) temporal convolution; *mt''*, middle (or second) temporal convolution; *ut'''*, inferior (or third) temporal convolution; *oo'*, superior (or first) occipital convolution; *mo''*, middle (or second) occipital convolution; *uo'''*, inferior (or third) occipital convolution; *op*, operculum; *osf*, superior frontal sulcus; *usf*, inferior frontal sulcus; *fs*, fissure of Sylvius, with its horizontal ramus on the right and its vertical ramus on the left; *cf*, central sulcus (fissure of Rolando); *cm*, callosomarginal fissure; *sip*, interparietal fissure; *fpo*, parieto-occipital or occipital fissure; *sf*, superior (or first) and inferior (or second) temporal fissures; *spc*, vertical frontal (precentral) fissure; *sot*, transverse occipital sulcus; *soli*, inferior longitudinal occipital sulcus.

The presence of *general* or *diffuse cerebral symptoms* answers the first question in the affirmative; *local* or *focal cerebral symptoms* furnish the answer to the second question; and the *clinical course of the disease* affords the basis for an answer to the third question.

General or *diffuse cerebral symptoms* appear in connection with all possible diseases of the brain without reference to the situation and anatomic character of the latter. They result from alterations in the circulation and the pressure within the cranium, and they include headache, vertigo, vomiting, disorders of consciousness, general clonic convulsions, choked disc, ringing in the ears, changes in the rhythm and the frequency of the pulse and in the respiratory movements, and the like.

The *local* or *focal symptoms* vary with the seat of the disease in the individual case. Naturally, there are as yet numerous *dead or silent points* in the brain; that is, regions that may be destroyed without giving rise to distinctive symptoms, if to any. Among the focal symptoms a distinction must be made between those that are *direct* and those that are *indirect.* Direct focal symptoms depend upon destruction of a portion of the brain directly, and are persistent, as regeneration of brain-tissue does not take place. They are, therefore, designated *symptoms of deficiency.* The indirect focal symptoms are known also as *remote symptoms,* as they are due to changes in pressure and circulation induced by a remotely situated lesion upon a number of otherwise uninjured portions of the brain. If the alterations referred to disappear, the indirect focal symptoms also may subside. In the presence of recent disease the distinction between direct and indirect focal symptoms can scarcely ever be made with certainty. Only those phenomena that persist after the lapse of two months generally constitute the direct focal symptoms or symptoms of deficiency. As much of existing knowledge concerning focal symptoms as can be utilized in diagnosis will be indicated in the following.

FOCAL SYMPTOMS ATTENDING DISEASE OF THE CEREBRAL CORTEX.

Upon the cerebral cortex *disease of the anterior and posterior central convolutions,* or, as they are sometimes also designated, the corticomuscular cortical region, is of especial importance. From the famous experiments on animals made by Hitzig, and which have been fully confirmed by observations on human beings, it is known that the central convolutions contain definite areas—*motor cortical centers*—irritation of which excites spasm, and destruction paralysis of definite groups of muscles and extremities upon the opposite side of the body. In the upper third of the anterior and the upper two-thirds of the posterior central convolution is situated the *center for the leg* (p. 31, Fig. 8), which upon the median aspect of the brain includes also the paracentral lobule (p. 31, Fig. 7). In the middle third especially of the anterior central convolution is situated the *arm-center* (Fig. 8). In the lower third of the anterior and posterior central convolutions the *facial*

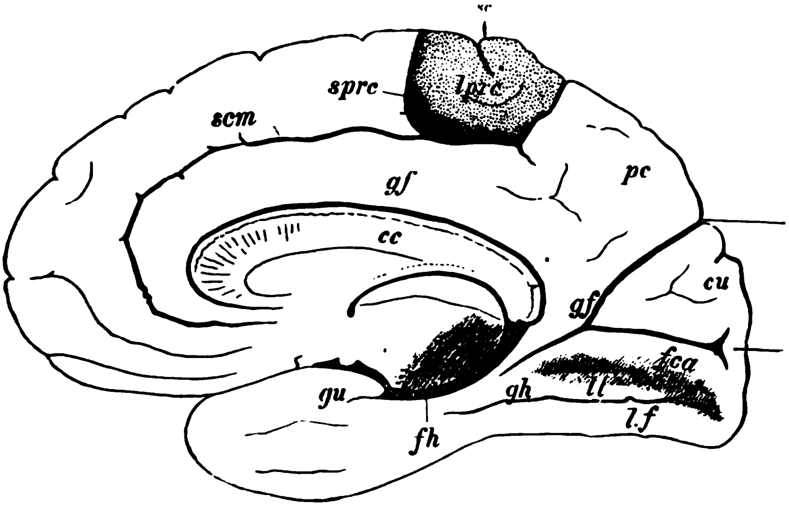


Fig. 7.—Median surface of the cerebral hemisphere (after Ecker): *lprc*, **paracentral lobule (leg-center)**; *sprc*, **paracentral sulcus**; *sc*, extremity of the central sulcus; *scm*, **callosomarginal sulcus**; *pc*, **precuneus**; *cu*, cuneus; *cc*, corpus callosum; *gu*, uncinate **gyrus**; *gh*, **hippocampal gyrus**; *gf*, fornicate gyrus; *gd*, descending gyrus; *ll*, **lingual lobule or lateral** occipitotemporal **gyrus**; *lf*, fusiform lobule or median occipitotemporal **gyrus**; *fpo*, **parieto-occipital** or occipital fissure; *fca*, calcarine fissure; *fh*, hippocampal **fissure.**

center is encountered; and finally in the lowest portion of the so-called opercular area is the *hypoglossal center* (Fig. 8).

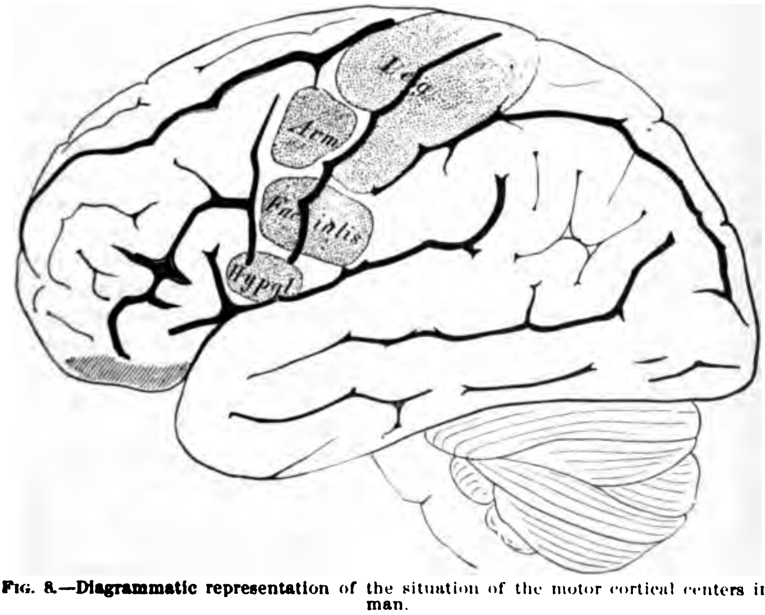


Fig. 8.—Diagrammatic representation of the situation of the motor cortical centers in man.

Within the individual motor cortical centers smaller areas can be delimited for individual muscle-groups, whose situation in man also is known in part, because in operations upon the brain the surgeon is at times compelled to stimulate the cerebral cortex of the living human being with small electrodes of a faradic current in order to assure himself of the relations of the parts. The anterior and posterior central convolutions can be readily recognized upon the cerebral cortex by finding the two branches of the *Sylvian fissure* at the base of the brain. In the angle formed by the junction of these fissures the *central sulcus* or *fissure of Rolando* passes from the median fissure. In front of this is the anterior and behind it the posterior central convolution, which unite below in the *lid* or *operculum* (p. 29, Fig. 6).

Microscopic examination of the cerebral cortex shows that in the cortical motor centers are situated large ganglion-cells, which on account of their shape are designated *pyramidal cells*. These cells must be considered as the beginning of the motor or corticomuscular pyramidal tract. Each of these cells sends out an axis-cylinder process, which without division passes through the corona radiata, the internal capsule, the cerebral peduncle, the pons, and the medulla oblongata, to enter either the anterior pyramidal tract on the same side, or, after crossing over in the pyramidal decussation, the lateral pyramidal tract of the opposite side of the spinal cord. From this situation it enters the anterior gray matter of the spinal cord, and gives off numerous branches, which apply themselves to the branches of the large motor-trophic ganglion-cells in the anterior horns of the spinal cord, in order to convey motor impulses to the latter. Each pyramidal cell, with its axis-cylinder, to the spinal ramification, is designated a *cerebrospinal neuron* or a *neuron of the second degree*.

Paralysis and muscular twitching naturally occur if the motor pyramidal tract is interrupted or is irritated at any point in its course. In order to determine whether a paralysis is of cortical origin the following facts should be borne in mind:

Cortical paralysis not rarely occurs in the form of *monoplegia*, because the lesion has destroyed the functional activity of but a single cortical center. It is distinguished as *central* monoplegia from *peripheral* monoplegia by an absence of any alteration in the electric irritability and of degenerative muscular atrophy. On the other hand, muscular contractures and exaggeration of the tendon-reflexes occur after a time, perhaps in consequence of secondary degeneration of the related pyramidal tracts.

In the course of diseases with a tendency to extension a cortical monoplegia may gradually be transformed into a cortical hemiplegia. *Gradual development of hemiplegia* is therefore indicative of cortical origin.

The mode of development of cortical hemiplegia is of diagnostic importance, because immediately adjacent cortical centers are involved successively. If, therefore, the arm-center is first attacked, involvement of the leg-center or of the facial center will next be superadded, and not that of the hypoglossal center, because it cannot readily be conceived that the facial center should thus escape.

Cortical paralysis is not rarely attended with *clonic spasm*, less commonly with tonic spasm; and this is explained by assuming that not all of the pyramidal cells of a center are destroyed, so that those that remain are irritated by the disease-focus and give rise to muscular contractions in the paralyzed members.

Cortical lesions that cause only irritation of the motor centers also induce only clonic muscular spasm. The cortical origin of such motor disturbances is indicated by persistent twitching of the same extremity. If, however, the clonic muscular contractions extend to adjacent motor centers—and this may be due to the remote action of even small foci of disease—the extension takes place in accordance with the anatomic relations of the individual centers, and intervening centers do not escape. In spite of general clonic muscular contractions consciousness is usually preserved. Under such circumstances the condition has been designated *cortical* or *Jacksonian epilepsy*.

The *cortex of the frontal portion of the brain* is associated with the processes of *thought* and of *intelligence*. In idiots the convolutions of the frontal region have been found but imperfectly developed. Perhaps disease of the gyrus rectus at the base of the brain is associated with *alterations in character*, and these may manifest themselves especially in a tendency to acts of violence. Lesions at the *foot of the lowermost left frontal convolution*, also known as the opercular portion of the inferior frontal convolution or as Broca's convolution, are of especial significance, as they cause *motor* (*ataxic*) *aphasia*, which will be more fully discussed subsequently.

Little of a definite nature is known with regard to the functions of the *cortex of the parietal region*. It is considered to be the cortical center for the *muscular sense*, so that patients whose cortex is destroyed in the parietal region have lost the power of determining the position of the extremities upon the opposite side of the body when the eyes are closed.

The *cortex of the left temporal lobe*, more accurately the posterior portion of the superior temporal convolution, contains the cortical radiation of the auditory nerve. Patients with destruction of this portion of the brain upon the left side suffer from *word-deafness* or *sensory aphasia*. Although they hear spoken words as sound, they are unable to comprehend and to utilize them. They are in the position of one hearing an unfamiliar and foreign language.

Injuries of the uncinate gyrus of the temporal portion of the brain (p. 31, Fig. 7) at the base of the brain are followed by disturbances in the sense of smell.

The *occipital portion of the brain*, finally, contains the *cortical radiations of the optic-nerve fibers*. Injuries of the cuneus and of the first occipital convolution (Fig. 7) are attended with *hemianopsia* (*hemiopia*).

The *occurrence of hemianopsia* may be explained as follows:

Within the chiasm of the optic nerves partial decussation of the fibers of these nerves takes place. At the same time the temporal portion of each

tract remains uncrossed, and only the fibers upon the nasal side undergo decussation (Fig. 9). Thus, for instance, the left optic tract supplies the temporal half of the left and the nasal half of the right retina. If, now, its cortical center in the left occipital lobe is destroyed, the power of vision is lost in the portions of the retina named, and the patient is incapable of seeing bodies brought toward him from the right. At times patients with injuries of the occipital cortex suffer from *mind-blindness*. They see objects, but do not recognize them, and they are incapable of comprehending what they see. If writing be not recognized, the condition is also spoken of as *word-blindness*. These conditions are associated with disease of the remainder of the occipital cortex with the exception of the cuneus.

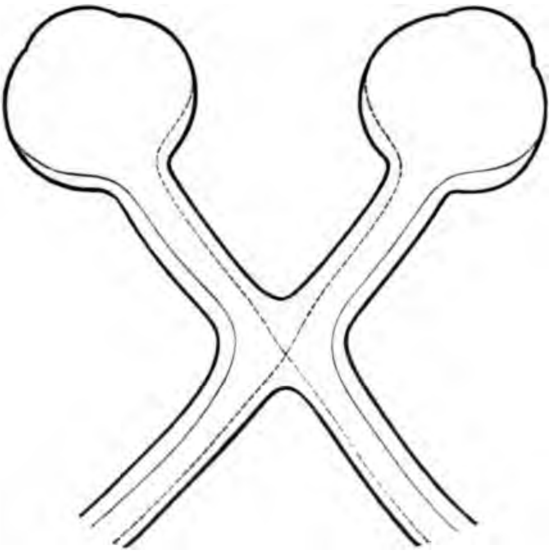


FIG. 9.—Diagrammatic representation of the decussation of the optic fibers in the chiasm.

FOCAL SYMPTOMS ATTENDING DISEASE OF THE CENTRUM SEMI-OVALE.

Situated between the cerebral cortex and the basal ganglia of the brain are white medullated masses, known as the centrum semi-ovale. These contain *association-fibers*, which connect points in the cerebral cortex of the same hemisphere; *commissural fibers*, which, through the corpus callosum, connect symmetrical points in the cerebral cortex of each hemisphere; and fibers of the *corona radiata*. The latter includes those systems of fibers that penetrate from the cerebral cortex into the depth of the brain and establish a connection between the cortex and the surface of the body. As yet, no disturbances are known that indicate disease of the association-fibers and the commissural fibers. If, however, a lesion occurs in the corona radiata, the same symptoms appear as if the lesion

**were situated in the related cortical area itself, and it is im
to determine with certainty during life whether a cortical
is present or one in the corona radiata. Foci of the latt**

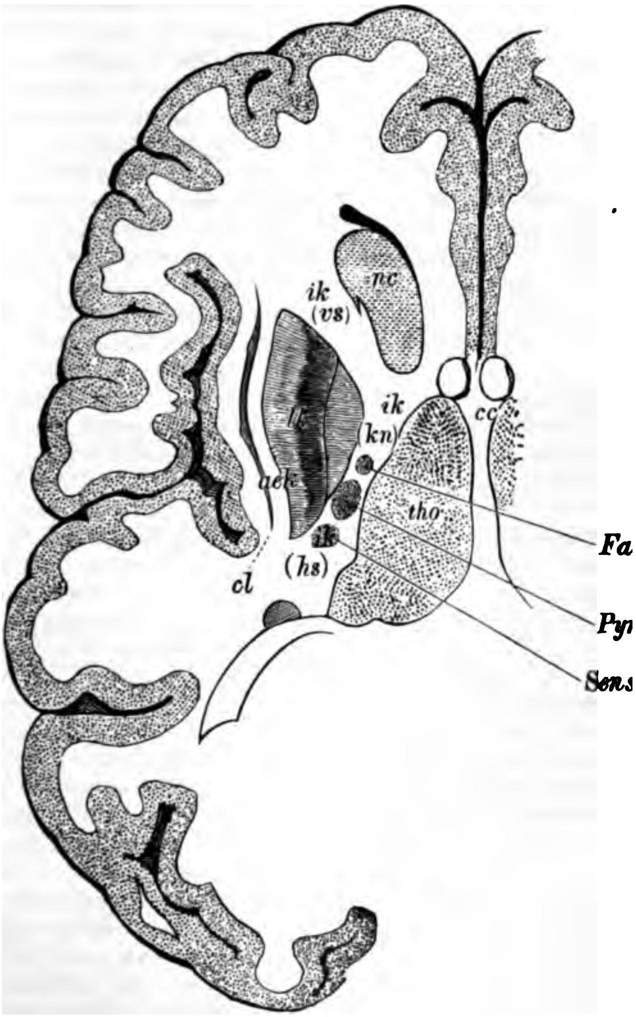


Fig. 10.—Horizontal section through the cerebrum : *ik*, internal capsule ; *(vs)*
limb ; (*hs*), posterior limb ; (*kn*), knee ; *lk*, lenticular nucleus, with its three divi
caudate nucleus ; *tho*, optic thalamus : *cc*, mammillary bodies : *aek*, external ca
claustrum ; *Fac*, facial fibers in the posterior limb of the internal capsule ; *Pyr*,
fibers for the extremities ; *Sens*, sensory tract.

are, however, extremely rare. Lesions of the corona radia
the central convolutions give rise to paralysis, and lesions
from the occipital region to hemianopsia, etc.

FOCAL SYMPTOMS ATTENDING DISEASE OF THE INTERNAL CAPSULE.

The internal capsule is the white medullary mass lying to the median aspect of the optic thalamus and the caudate nucleus, and to the lateral aspect of the lenticular nucleus (p. 35, Fig. 10). It presents an anterior and a posterior limb, which join to form the knee of the internal capsule. The anterior limb is yet included among the silent portions of the brain. Through the posterior limb, however, the motor pyramidal tract, as well as the sensory and the sensorial paths, pursues its course. If the posterior limb is divided into three equal parts, the pyramidal tract will be situated in the middle third. In the most anterior portion of this is situated the path for the facial nerve, and close to this that for the hypoglossal nerve. Then follows the path for the arm, and finally that for the leg. Hemorrhage into the internal capsule is one of the commonest of cerebral disorders. If the middle third of the posterior limb should be involved, the fact will be disclosed by paralysis of the facial and hypoglossal nerves and of the arm and the leg upon the opposite side of the body. The condition is then designated *ordinary cerebral hemiplegia.* The facial palsy, however, is unattended with involvement of the frontal branch for the frontal and orbicular muscles. Destructive lesions in the posterior third of the posterior limb of the internal capsule are followed by *cerebral cutaneous anesthesia.* The patient will then have lost the sense of touch upon the opposite half of the body, and often also the special senses (vision, smell, taste).

The ganglia at the base of the brain, the *caudate nucleus* and the *lenticular nucleus,* together known as the *striate body,* are as yet included among the silent areas of the brain.

FOCAL SYMPTOMS ATTENDING DISEASE OF THE CEREBRAL PEDUNCLES.

Lesions in the cerebral peduncles can be recognized from the presence of *cerebral hemiplegia* with *alternate oculomotor paralysis;* that is, the facial and hypoglossal nerves and the arm and the leg are paralyzed upon the side opposite that of the lesion, while the oculomotor is paralyzed upon the same side as the lesion.

On *transverse section through the cerebral peduncle* several layers may be distinguished. Toward the ventral aspect is the *foot of the cerebral peduncle.* Above it is the *substantia nigra,* the brownish color of which is due to the large amount of pigment contained within its ganglion-cells; and uppermost is the *tegmentum* (Fig. 11). Motor paralysis results from lesions in the cerebral peduncle if these be situated in the middle third, for the motor pyramidal tract descends through this part to the pons and below, the path for the facial nerve being farthest toward the median aspect, to its outer side the fibers of the hypoglossal, and next those for the arms and the legs. The paralysis of the structures named occurs upon the side of the body opposite to the lesion, because the nerve-tracts in question undergo decussation at a lower level and pass to the opposite side of the body. Involvement of the oculomotor nerve results because this nerve traverses the cerebral peduncle in order to reach its nucleus at the side of the aqueduct of Sylvius. The oculomotor palsy must therefore occur upon the same side as the lesion, for it is to a certain degree peripheral in character and engendered within the intramedullary course of the nerve to the peripheral aspect of its decussation. Alternating oculomotor palsy, however, occurs only as the result of lesions of the cerebral peduncle if these are situated in the spinal (posterior) half of the peduncle. Lesions in the cerebral (anterior) portion give rise only to simple cerebral hemiplegia, and the oculomotor nerve remains uninvolved, as its course does not traverse the anterior portion of the cerebral peduncle. The paralysis, therefore, wholly resembles that due to capsular lesions, from which it is indistinguishable. *Disease of the tegmentum* gives rise to cerebral hemianesthesia because the sensory tracts are contained in the tegmentum.

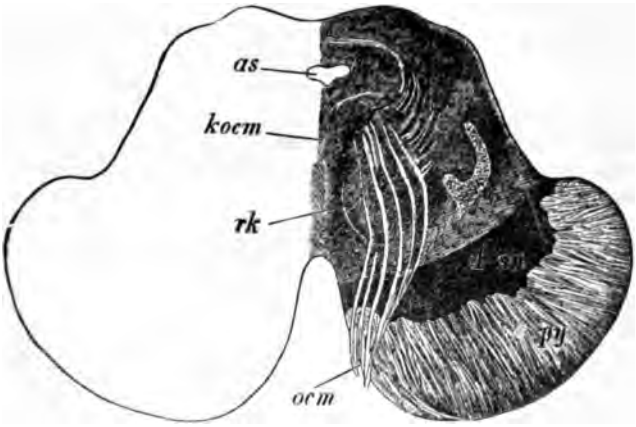


FIG. 11.—Transverse section through the cerebral peduncle corresponding with the posterior portion of the anterior quadrigeminal body: *ocm*, oculomotor nerve; *kocm*, nucleus of the oculomotor nerve; *py*, pyramidal tract; *sn*, substantia nigra; *rk*, red nucleus; *as*, aqueduct of Sylvius. Enlarged three times.

FOCAL SYMPTOMS ATTENDING DISEASE OF THE PONS.

Disease of the posterior (spinal) portion of the pons Varolii must always be thought of in the presence of *hemiplegia with alternate facial paralysis*. Under such circumstances the arm and the leg are paralyzed upon the side opposite to that of the lesion, the facial nerve, however, upon the same side as the lesion. The conditions here are the same as have been described for lesions of the cerebral peduncle with regard to the oculomotor nerve. The trunk of the facial nerve traverses the posterior half of the pons, reaches its nucleus in the depth of the floor of the fourth ventricle, and undergoes decussation to the cerebral aspect of the nucleus, finally to reach its cortical center in the opposite cerebral hemisphere. Lesions to the peripheral aspect of the decussation must, therefore, give rise to facial palsy of a peripheral character upon the same side, while the arm and the leg are paralyzed upon the opposite side of the body, as their paths cross over in the pyramidal decussation at a lower level. It is distinctive, further, of this variety of facial paralysis that the frontal branch also is involved, and that degenerative electric reaction occurs in the paralyzed facial muscles.

Lesions in the anterior (*cerebral*) *portion of the pons* are attended with ordinary cerebral hemiplegia, and under such circumstances pontine symptoms must always be looked for in order to make a correct local diagnosis. Among pontine symptoms may be mentioned irritative and paralytic phenomena involving certain cerebral nerves whose nuclei and intramedullary paths are situated in the pons. Frequently the nerve-nuclei in the medulla oblongata also are involved, and these cannot wholly be separated from those of the pons. *Pontine symptoms* include trismus (from irritation of the motor trigeminal segment), unusually small pupils (myosis), exceedingly high bodily temperature, together with difficulty in swallowing, anesthesia, increased secretion of saliva, increased pulse-frequency, polyuria, and glycosuria. Often clonic muscular spasm is observed, and this has been attributed to irritation of a hypothetic spasm-center in the pons. Further, symptoms of *paralysis of cerebral nerves of pontine origin* alone occur at times, and these, accordingly as the lesion is situated to the cerebral or the spinal aspect of the decussation, may be present now upon the opposite and again upon the same side of the body. Also the motor pyramidal tracts are at times only partially affected, so that *pontine monoplegia* may be present.

Sometimes *conjugate deviation of the eyes and the head* has been observed in conjunction with pontine lesions, the eyes and the head being deflected from the lesion. This symptom is, however, of diagnostic significance only when it is due to paralysis of ocular muscles (abducens), and not to irritation.

Disease of the optic thalamus can but rarely be diagnosed. Involvement of the posterior portion (pulvinar, external geniculate body) is attended with *homonymous* (*lateral*) *hemianopsia.* Destructive lesions in the anterior portion are said to suppress *mimic movements of the face*, while the voluntary movements of the facial muscles are preserved. *Hemichorea* and *athetosis* also have been observed in association with lesions of the optic thalamus, although these conditions occur only when the lesion in the optic thalamus exerts irritation of the adjacent motor pyramidal tract in the internal capsule. Lesions in the *external capsule*, the *claustrum*, and the *cornu ammonis* are unattended with distinctive symptoms.

The *quadrigeminate bodies* are endowed with several functions. Lesions in the anterior quadrigeminate body, if unilateral, are attended with *hemianopsia*, while bilateral lesions are attended with *amblyopia* or *amaurosis*. Lesions of the posterior quadrigeminate body give rise to *oculomotor paralysis*, and at times also to trochlear paralysis. The symptoms of *cerebellar ataxia* also are attributed to such lesions.

No distinctive symptoms are known to result from *disease of the cerebellum*. Possibly involvement of the vermis gives rise to *vertigo* and *stagger-*

ing gait (*cerebellar ataxia*). *Disease of the cerebellar peduncles* is usually unrecognized. At times lesions of the *middle cerebellar peduncle* are attended with *rotatory movements of the head about its longitudinal axis*, sometimes toward the side of the lesion, and at other times toward the opposite side, but only when the lesion gives rise to irritative effects.

No focal symptoms of *lesions of the corpus callosum* are known, although apathy, somnolence, and disorders of equilibration and in the more delicate synergistic movements of the body have been mentioned as such.

FOCAL SYMPTOMS ATTENDING DISEASE AT THE BASE OF THE BRAIN.

Lesions on the floor of the cranium and at the base of the brain are not uncommon, and they are associated with especial frequency with inflammatory thickening or neoplasms of the meninges. Under such conditions cerebral nerves are readily compressed and paralyzed. The nerves involved will naturally vary accordingly as the lesion is situated in the anterior, the middle, or the posterior fossa of the skull. Not rarely, lesions at the base of the brain extend progressively and involve a larger and larger number of cerebral nerves. The accompanying illustration will aid in a comprehension of the symptoms (p. 41, Fig. 12).

THE SYMPTOM-COMPLEX APHASIA.

Certain disturbances of speech designated *aphasia* must likewise be included among the focal symptoms of disease of the brain, as they bespeak disease of a definite portion of the cerebral cortex. It is noteworthy, in the first place, that aphasic disorders occur only in connection with *disease of the left cerebral hemisphere*, obviously because we have become accustomed to educate and to employ the left hemisphere in the processes of speech. Only left-handed persons constitute an exception to this rule; in them the right cerebral hemisphere preponderates over the left.

Three principal varieties of aphasia may be distinguished, and these have been designated motor (ataxic), sensory (word-deafness), and amnesic. *Motor* or *ataxic aphasia* occurs when the foot (*opercular portion*, Broca's area) of the *lowermost or third left frontal convolution* is destroyed. This is the region with which the cortical centers for the hypoglossal nerve and the muscles of the larynx stand in most intimate relation. Motor aphasia is characterized by an impossibility of orderly and coördinated action of the muscles of speech in spite of preservation of the power of articulation, so that words are imperfectly enunciated on attempted repetition and in spontaneous speech. In accordance with the severity of the disease the degree of motor aphasia likewise is quite variable. In the mildest cases close attention will be required in order to recognize at all the distortion of words, the confusion of letters, while in severe cases patients may be therefore, give rise to facial palsy of a peripheral character upon the same side, while the arm and the leg are paralyzed upon the opposite side of the body, as their paths cross over in the pyramidal decussation at a lower level. It is distinctive, further, of this variety of facial paralysis that the frontal branch also is involved, and that degenerative electric reaction occurs in the paralyzed facial muscles.

Lesions in the anterior (cerebral) portion of the pons are attended with ordinary cerebral hemiplegia, and under such circumstances pontine symptoms must always be looked for in order to make a correct local diagnosis. Among pontine symptoms may be mentioned irritative and paralytic phenomena involving certain cerebral nerves whose nuclei and intramedullary paths are situated in the pons. Frequently the nerve-nuclei in the medulla oblongata also are involved, and these cannot wholly be separated from those of the pons. *Pontine symptoms* include trismus (from irritation of the motor trigeminal segment), unusually small pupils (myosis), exceedingly high bodily temperature, together with difficulty in swallowing, anesthesia, increased secretion of saliva, increased pulse-frequency, polyuria, and glycosuria. Often clonic muscular spasm is observed, and this has been attributed to irritation of a hypothetic spasm-center in the pons. Further, symptoms of *paralysis of cerebral nerves of pontine origin* alone occur at times, and these, accordingly as the lesion is situated to the cerebral or the spinal aspect of the decussation, may be present now upon the opposite and again upon the same side of the body. Also the motor pyramidal tracts are at times only partially affected, so that *pontine monoplegia* may be present.

Sometimes *conjugate deviation of the eyes and the head* has been observed in conjunction with pontine lesions, the eyes and the head being deflected from the lesion. This symptom is, however, of diagnostic significance only when it is due to paralysis of ocular muscles (abducens), and not to irritation.

Disease of the optic thalamus can but rarely be diagnosed. Involvement of the posterior portion (pulvinar, external geniculate body) is attended with *homonymous (lateral) hemianopsia.* Destructive lesions in the anterior portion are said to suppress *mimic movements of the face*, while the voluntary movements of the facial muscles are preserved. *Hemichorea* and *athetosis* also have been observed in association with lesions of the optic thalamus, although these conditions occur only when the lesion in the optic thalamus exerts irritation of the adjacent motor pyramidal tract in the internal capsule. Lesions in the *external capsule*, the *claustrum*, and the *cornu ammonis* are unattended with distinctive symptoms.

The *quadrigeminate bodies* are endowed with several functions. Lesions in the anterior quadrigeminate body, if unilateral, are attended with *hemianopsia*, while bilateral lesions are attended with *amblyopia* or *amaurosis*. Lesions of the posterior quadrigeminate body give rise to *oculomotor paralysis*, and at times also to trochlear paralysis. The symptoms of *cerebellar ataxia* also are attributed to such lesions.

No distinctive symptoms are known to result from *disease of the cerebellum*. Possibly involvement of the vermis gives rise to *vertigo* and *stagger-*

[illegible] *muscle* [illegible] effects.

No focal symptoms [illegible] although [illegible] disorders [illegible] movements [illegible]

FOCAL SYMPTOMS ATTENDING DISEASE AT THE BASE OF THE BRAIN

Lesions of the floor of the cranium and at the base [illegible] are not uncommon, and they are associated with [illegible] with inflammatory thickening of the [illegible] Under such conditions cranial nerves are [illegible] and paralyzed. The nerves involved will naturally vary accordingly as the lesion is situated in the anterior, the [illegible] or the posterior fossa of the skull. Not rarely, lesions at the [illegible] brain extend progressively and involve a [illegible] ber of cranial nerves. The accompanying [illegible] in a comprehension of the symptoms [illegible]

THE SYMPTOM-COMPLEX APHASIA

Certain disturbances of speech designated [illegible] wise be included among the focal symptoms [illegible] brain, as they represent disease of a [illegible] cortex. It is noteworthy, in the first place [illegible] occur only in connection with [illegible] obviously because we have become accustomed [illegible] employ the left hemisphere in the process of speech [illegible] handed persons constitute an exception to this [illegible] right cerebral hemisphere [illegible]

Three principal varieties [illegible] and these have been designated [illegible] ataxic [illegible] ness, and amnesic. *Motor* [illegible] the foot [illegible] *portion*. Broca's [illegible] *frontal convolution* is destroyed. [illegible] which the cortical centers for the [illegible] of the larynx cannot in most [illegible] aphasia is characterized by an impossi[illegible] coordinated action of the muscles of speech [illegible] of the power of articulation, so that [illegible] enunciated on attempted repetition and [illegible] In accordance with the severity of the [illegible] aphasia likewise is quite variable. [illegible] attention will be required in order to [illegible] of words, the confusion of letters, whi[illegible] may be scarcely able to enunciate remnants or fragments of words. In the latter event it not rarely happens that, in their efforts to speak, the patients often use the same words, phrases, or exclamations, and these often are senseless—so-called *monophasia.*

Sensory aphasia or *word-deafness* depends upon *disease of the posterior half of the superior (first) temporal convolution.* It can be recognized from the failure of the patient to comply with requests, such as extension of the hand, closure of the eyes, elevation of the leg, protrusion of the tongue, because the patient, although he perceives words as sounds, does not grasp the sense or the significance of the words. He is in the position of being spoken to in a foreign language. In examining the patient care should be taken that in making a request this is not accompanied by expressive gestures or facial expression, for it may happen that although the patient does not understand the words he apprehends the gestures or the facial expression, and responds thereto. It should be recalled that the temporal convolutions are considered as the cortical area for the radiations of the auditory nerve.

Amnesic aphasia is attended with failure to recall words. If articles are held before the patient or if familiar objects are pointed out (eye, nose, mouth, finger), he will be unable to indicate them by name. Even when he desires to designate these things spontaneously words will fail him. The patient has at times forgotten his own name. Instead of the word desired, others are not rarely spoken—so-called *paraphasia.* Whether the seat of amnesic aphasia is to be looked for in the *convolutions of the island of Reil* appears doubtful.

In persons who have been familiar with *several languages* it has occasionally been observed that they suffer from amnesic aphasia with particular reference to one language, and at times especially the mother-tongue. Further, in addition to the three principal varieties of aphasia named several other varieties occur. Thus, the disturbance of speech as a result of which patients are unable to designate by name objects seen, although they are able to name the object when it is palpated, has been called *optic aphasia.* There is also a *tactile* and an *auditory* variety of aphasia. Often aphasia appears to be dependent upon the fact that the patients are unable to retain for a sufficient length of time the memory of conceptions of sound, vision, and hearing.

Clinically a distinction must be made between *simple* and *mixed aphasia,* accordingly as the symptoms of one variety of aphasia or of several are present at the same time. Most commonly the one variety predominates, while indications of other varieties are present at the same time. As the portions of the brain concerned have a common arterial blood-supply, namely, the middle cerebral artery or the artery of the fissure of Sylvius, it can be understood that emboli or thrombi in this vessel may readily involve several cerebral convolutions, and accordingly may give rise to a mixed variety of aphasia (p. 42, Fig. 13).

In conjunction with disorders of speech difficulty in reading, in writing, and in communication by gesture may occur; and these conditions are designated respectively *alexia*, *agraphia*, and *amimia.* All of these disorders together constitute the aphasic symptom-complex. At times the patient has lost all power of communicating with his fellows by writing, by speech, or by gesture, and of understanding others through speech, writing, and gestu

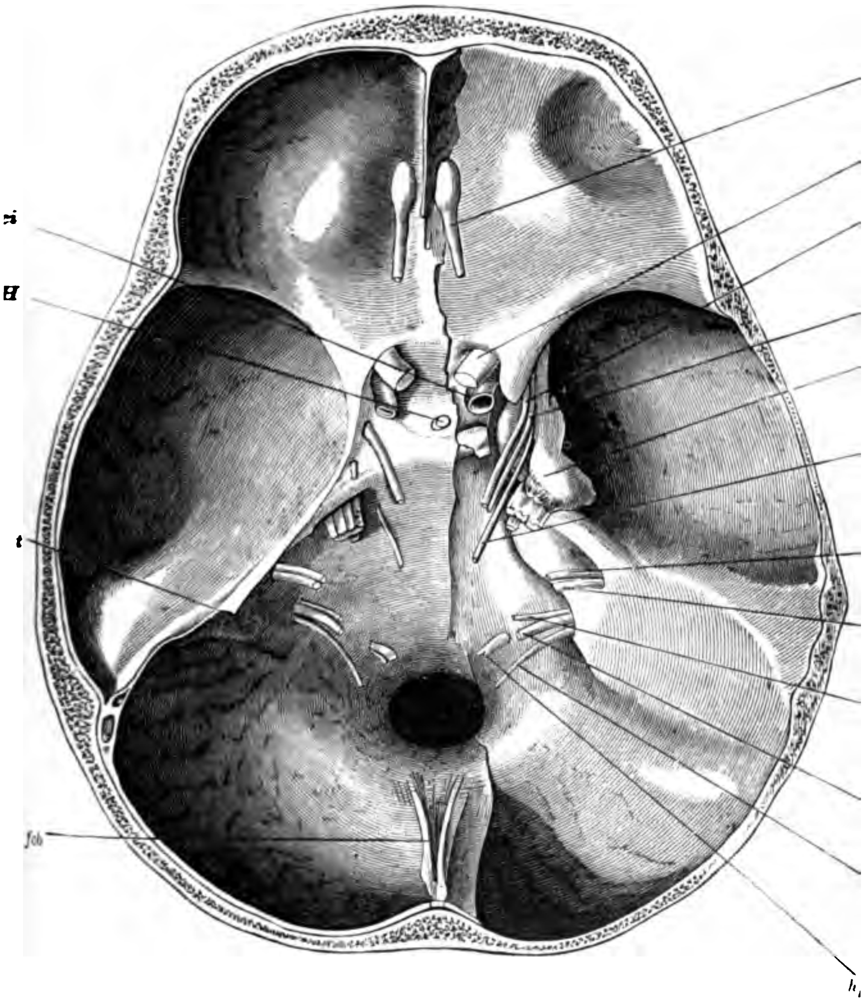


FIG. 12.—Floor of the skull showing the exit of the cerebral nerves (after Henle): *Ol*, olfactory nerve; *Op*, optic nerve; *ocm*, oculomotor nerve; *tr*, trochlear nerve; *trg*, trigeminal nerve; *ab*, abducens nerve; *fc*, facial nerve; *ac*, auditory nerve; *gl*, glossopharyngeal nerve; *vg*, vagus; *acc*, accessory nerve; *hp*, hypoglossal nerve; *ci*, internal carotid artery; *H*, hypophysis; *t*, apex of the tentorium; *fcb*, falx cerebelli.

The patient has lost his *facultas signatrix*, and suffers from *asymbolia* or *asemia*. Patients with motor aphasia naturally exhibit also *motor alexia*. Sensory aphasia bespeaks *sensory alexia*, in the presence of which the patients do not comprehend the sense of what is read. They therefore fail to comply with written requests. *Motor agraphia* renders the patient incapable of writing by transcript, while in the presence of *amnesic agraphia* that which is thought and willed cannot be recorded on paper. Inability to write from dictation would be designated *sensory agraphia*.

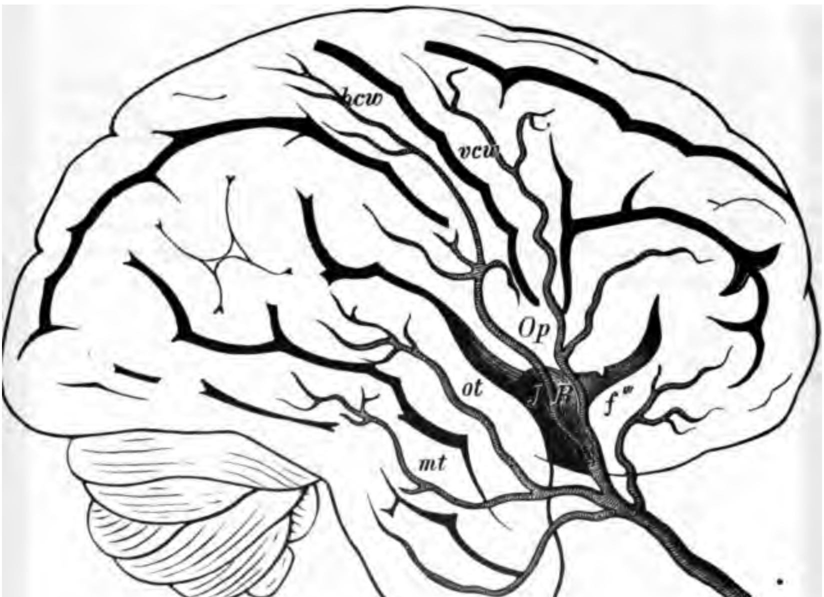


FIG. 13.—Distribution of the artery of the fossa of Sylvius upon the cerebral cortex, partially diagrammatic: *Art.f.S*, artery of the fossa of Sylvius; *f'''*, inferior frontal convolution; *Op*, operculum; *vcw*, anterior central convolution; *hcw*, posterior central convolution; *ot*, superior temporal convolution; *mt*, middle temporal convolution; *JR*, island of Reil.

Disorders occur in the *mimic field* in quite a corresponding manner. Gestures are not imitated—*motor amimia*—or are not understood—*sensory amimia*—or they cannot be executed—*amnesic amimia*.

The aphasic symptom-complex is by no means entirely exhausted by the alterations described. Analogous disturbances may, for instance, occur in the *musical faculty*, so that the patients are unable to repeat well-known melodies sung to them, or are unable to recognize them; or they are unable to sing a melody with which they had previously been familiar. *Disorders of the power of calculation* have also been observed, and the like. At times the patients have lost their comprehension of the use of objects and suffer from *apraxia*. If they be given some article of clothing, they are incapable of deciding what to do with it. A candle may, for instance, be used for washing the hands, while soap may be introduced into the mouth, and the like.

The doctrine of aphasia, which was first established especially by two French physicians, Dax and Broca, has been subjected to most assiduous investigation up to the present time. Various *diagrams* have been constructed for the purpose of explaining the mechanism of the aphasic symptom-complex, but some cases will not fit into such explanations; so that a number of problems still remain to be solved in this connection. The strictly aphasic disturbances can be readily comprehended from the accompanying diagrams (Figs. 14 and 15). In order to acquire the power of speech as a child, it is necessary for the individual to hear spoken words and to attempt their reproduction. Accordingly, the path for the auditory nerve, the cortical auditory center in the superior temporal convolution, the path of communication with the cortical center for the muscles of speech in the inferior frontal convolution, and the peripheral path for the speech-muscles must be intact (Fig. 14). A child first repeats words without comprehending their significance, and only gradually it learns to associate a definite conception or idea with each word. This can take place only by the establishment of a path of communication between the cortical auditory center and the concept-center, and also of a path of communication between the latter and the cortical center for the muscles of speech. The latter is necessary for spontaneous, intelligent speech. Fig. 15 represents a *diagram for the comprehension of intelligent speech.*

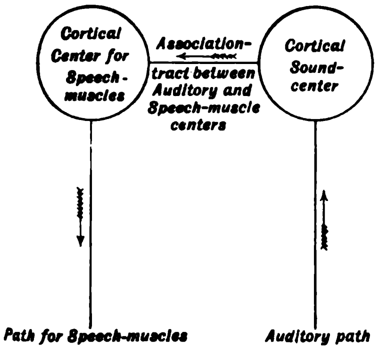

Cortical Center for Speech-muscles
Association-tract between Auditory and Speech-muscle centers
Cortical Sound-center
Path for Speech-muscles
Auditory path

FIG. 14.—Diagram illustrating the processes in the acquisition of speech.

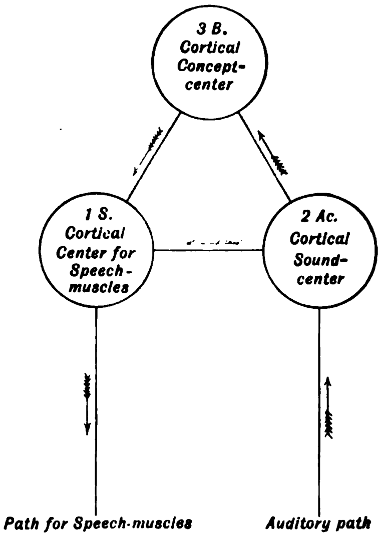

3 B. Cortical Concept-center
1 S. Cortical Center for Speech-muscles
2 Ac. Cortical Sound-center
Path for Speech-muscles
Auditory path

FIG. 15.—Diagram illustrating the processes of acquired speech.

Disorders of speech, or aphasia, will occur either if the cortical auditory center or the cortical center for the muscles of speech is destroyed—*cortical aphasia;* or if the paths of communication between the cortical auditory center or the center for the muscles of speech and the concept-center are interrupted—*transcortical aphasia;* or if the path for the auditory nerve or for the muscles of speech is injured—*subcortical aphasia;* or, finally, if an interruption has taken place in the conduction-path between the cortical auditory center and the cortical center for the muscles of speech—*conduction-aphasia.*

Destruction of the cortical auditory center causes sensory aphasia. The patient fails to comprehend spoken words, and is unable to repeat them. On the other hand, spontaneous speech is not interfered with. Naturally, improper words or words with a similar sound are often used in speech—so-called *paraphasia. Destruction of the path of communication between the cortical auditory center and the concept-center* gives rise to *transcortical sensory aphasia.* The patient fails likewise to comprehend spoken words, but is able to repeat them. Spontaneous speech is not interfered with, and is only modified by *paraphasia. Destruction of the path for the auditory nerve* gives rise to *subcortical sensory aphasia.* This entirely resembles cortical sensory aphasia (loss of the comprehension of words and inability to repeat spoken words), although, as will be explained later, other agraphic disturbances are present. *Destruction of the cortical center for the muscles of speech* induces *cortical motor* or *ataxic aphasia.* The patient, in spontaneous speech and on repetition, is capable, at best, of using distorted words. *Destruction of the path of communication between the cortical center for the muscles of speech and the concept-center* gives rise to *transcortical motor aph*[illegible] The patient comprehends spoken language, and is also capable o[illegible], while the

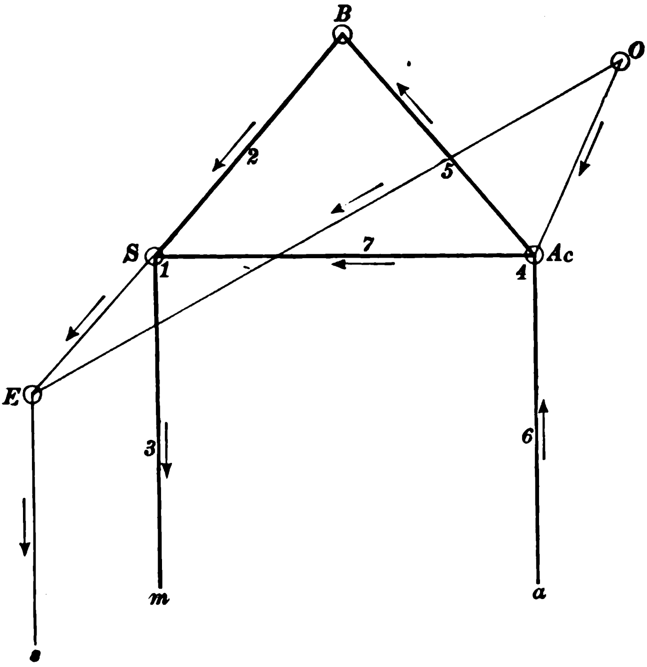


FIG. 16.—Diagrammatic representation of the entire aphasic symptom-complex: *B*, cortical concept-center; *S*, cortical center for the muscles of speech; *Ac*, cortical auditory center; *O*, cortical optic center; *E*, cortical center for the movements of writing; *aAc*, path for the auditory nerve; *mS*, path for the muscles of speech; *Es*, path for the movements of writing.

power of spontaneous speech is lost. ***Destruction of the path for the muscles of speech*** gives rise to ***subcortical motor aphasia.*** This closely resembles cortical motor aphasia (disturbance of the power of spontaneous speech and of repetition, with preservation of the comprehension of spoken language), although it is attended with other agraphic disturbances. Patients with subcortical motor aphasia are thought to be capable also of designating the number of syllables of which a spoken word is composed, for instance, through pressure with the hands, while this power is lost in the presence of cortical motor aphasia. *Destruction of the path of communication between the cortical auditory center and the center for the muscles of speech* gives rise to *conduction-aphasia.* The patient comprehends spoken language and can also speak spontaneously; but the *power of repetition* is interfered with. The disturbance may be mitigated through the intermediation of the concept-center, although there is frequently confusion of words—*paraphasia.*

For the comprehension of the alexic and agraphic disturbances that so frequently occur in association with aphasia, diagrams have been constructed that take into consideration the cortical optic centers and their paths of communication, and the cortical center for the movements of writing and their conduction-paths. Fig. 16 represents such a diagram, from which the aphasic symptom-complex may be comprehended in accordance with the seat of the disease.

1. *Cortical motor aphasia* (Fig. 16, 1).
    - ***Loss*** of the power of spontaneous speech.
    - Loss of the power of repeating spoken words.
    - Loss of the power of reading.
    - Loss of the power of writing from dictation.
    - Loss of the power of writing spontaneously.
    - Loss of the power to designate the number of syllables in words.
    - ***Preservation*** of the power of comprehending speech.
    - Preservation of the power of comprehending writing.
    - Preservation of the power of writing by transcript.

2. ***Transcortical motor aphasia*** (Fig. 16, 2).
    - ***Loss*** of the power of spontaneous speech.
    - Loss of the power of writing spontaneously.
    - ***Preservation*** of the power of comprehending speech.
    - Preservation of the power of repeating spoken words.
    - Preservation of the ability to read aloud.
    - Preservation of the power of comprehending writing.
    - Preservation of the ability to write by transcript.
    - Preservation of the ability to write from dictation.

3. ***Subcortical motor aphasia*** (Fig. 16, 3).
    - ***Loss*** of the power of spontaneous speech.
    - Loss of the power of repeating spoken words.
    - Loss of the ability to read aloud.
    - ***Preservation*** of the power of comprehending speech.
    - Preservation of the power of comprehending writing.
    - Preservation of the power of writing spontaneously.
    - Preservation of the ability to write by transcript.
    - Preservation of the ability to write from dictation.

4. ***Cortical sensory aphasia*** (*word-deafness*) (Fig. 16, 4).
    - ***Loss*** of the power of comprehending speech.
    - **Loss** of the power of repeating spoken words.
    - **Loss** of the ability to read.

Loss of the ability to write from dictation.
Loss of the ability to read aloud.
Preservation of the power of writing spontaneously.
Preservation of the ability to write by transcript.
Preservation of the power of speaking spontaneously.

5. *Transcortical sensory aphasia* (Fig. 16, 5).
    - *Loss* of the power of comprehending speech.
    - Loss of the power of comprehending writing.
    - *Preservation* of the power of spontaneous speech, with paraphasia.
    - Preservation of the power of repeating words, without ability to comprehend them.
    - Preservation of the ability to read aloud, without the power of comprehending words.
    - Preservation of the ability to write spontaneously, with paragraphia.
    - Preservation of the ability to write from dictation without the power of comprehension.
    - Preservation of the ability to write by transcript.
6. *Subcortical sensory aphasia* (Fig. 16, 6).
    - *Loss* of the power of comprehending speech.
    - Loss of the power of repeating words.
    - Loss of the ability to write from dictation.
    - *Preservation* of the power of speaking spontaneously.
    - Preservation of the ability to read aloud.
    - Preservation of the power of comprehending writing.
    - Preservation of the power of spontaneous writing.
    - Preservation of the ability to write by transcript.
7. *Conduction-aphasia* (Fig. 16, 7).
    - *Loss* of the power of repeating words.
    - Loss of the ability to read aloud.
    - Loss of the ability to write from dictation.
    - *Preservation* of the power of spontaneous speech, with paraphasia.
    - Preservation of the power of comprehending writing.
    - Preservation of the power of comprehending speech.
    - Preservation of the power of writing spontaneously, with paragraphia.
    - Preservation of the ability to write by transcript.

Most commonly *aphasia occurs in association with right-sided cerebral hemiplegia*. This is to be attributed to the fact that *emboli* and *thrombi*, the most common causes of aphasia, are, as a rule, situated in the middle cerebral artery or the artery of the fossa of Sylvius, so that, in addition to the third frontal and the first temporal convolution, also the two central convolutions, with their motor cortical centers, are deprived of their blood-supply (p. 42, Fig. 13). Aphasia without motor paralysis and pure varieties of aphasia occur only in association with the presence of small circumscribed lesions, as, for instance, tumors, abscesses, localized meningitis, and injuries. Thus, an observation is on record in which a man, after having been thrown from a horse, became suddenly aphasic, because a splinter, sprung from the vitreous plate of one of the cranial bones, lacerated the left third frontal convolution in Broca's area.

At times aphasic manifestations occur so abruptly as a transitory manifestation that *circulatory disturbances* in the speech-convolutions susceptible of being compensated for must be thought of. Such manifestations have been observed in cases of hysteria, epilepsy, uremia, and after infectious diseases and intoxications. Aphasia will disappear also, although more slowly, if it be not the result of a lesion of the speech-cortex, but is a *remote symptom* in the presence of hemorrhage, neoplasm, abscess, or an inflammatory process in the neighborhood of the speech-convolutions, and the pressure or analogous disturbance subside with the occurrence and the progress of absorption. *Congenital aphasia* has occasionally been observed. It is noteworthy that in a case of aphasia the brain may appear normal to the unaided eye, and only on *microscopic examination* will the changes in the cerebral cortex be disclosed.

Aphasic disturbances are susceptible of improvement, and occasionally of cure. These ends may be attained by *speech-exercises*. Patients suffering from motor aphasia should be made to repeat words, passing from the simple to the complex, and constantly repeating the same words until they are retained. Patients with amnesic aphasia are best educated by means of pictures. Naturally, much patience will be required on the part of both pupil and teacher. Care should be taken not to tire the patient, as aphasic individuals readily become impatient and angry. The greatest difficulty is encountered in the treatment of total aphasia—that is, simultaneous motor and sensory aphasia—as the means of communication with the patient are so limited. The possibility of learning to speak depends upon whether some parts of the speech-centers still persist, or whether the corresponding portions of the right cerebral hemisphere can be trained in the faculty of speech. Naturally, the primary disorder should be treated in accordance with the usual rules.

CEREBRAL ANEMIA.

Etiology.—Although the brain responds most delicately in function to all circulatory alterations, cerebral anemia but rarely acquires independent clinical significance. Cerebral anemia may, naturally, occur after **profuse loss of blood**, whether this takes place following injury or operation, or from the nose, the stomach, the intestine, the uterus, and the like. A **deficiency in the hemoglobin of the blood** exerts the same effect as hemorrhage, and symptoms of cerebral anemia are therefore observed in association with chlorosis, leukemia, pseudoleukemia, progressive pernicious anemia, the carcinomatous cachexia, long-continued diarrhea, and purulent and other wasting discharges. Cerebral anemia occurs not rarely in consequence of **spasm of the cerebral arteries.** Such an occurrence may be induced by an offensive visual impression, and even by a harrowing recital, by fright or pain, and then frequently causes **fainting—syncope.** Occasionally cerebral anemia is induced by the **sudden diversion of an excessive amount of blood to other organs.** Thus, it is observed at times after too rapid evacuation of peritoneal or pleural effusions. Also, the manifestations of shock following general concussion and traumatism are to be explained by paralysis of the splanchnic nerve, in consequence of which the blood-vessels of the abdominal viscera become greatly dilated and excessively filled with blood, while the brain is correspondingly deprived of blood. At times symptoms of cerebral anemia appear after a copious evacuation from the bowels that has for a long time been preceded by constipation, because the intestinal blood-vessels, previously compressed by the fecal accumulation, suddenly receive large amounts of blood after the expulsion of the contents of the bowel. Cerebral anemia may result from **cardiac weakness,** and is observed especially in conjunction with fat heart, because the heart is unable to propel a sufficient amount of blood into the brain. The disturbances generally occur in the upright posture. Symptoms of cerebral anemia appear not rarely also in association with stenosis of the aortic orifice. Cerebral anemia may develop in consequence of **increased cerebral pressure,** induced either from within outward (internal hydrocephalus) or being exerted from without inward (diffuse meningitis). Circumscribed cerebral anemia, such as occurs in connection with neoplasms, abscesses, hemorrhage, embolism and thrombosis of the cerebral arteries, may for the present be omitted from consideration.

Anatomic Alterations.—In the presence of cerebral anemia the brain-tissue is characterized by unusual **pallor.** Often the boundary between the white and the gray matter is ill defined. The brain-tissue is rather firm, providing that cerebral edema is not present together with cerebral anemia. The **meninges** also appear anemic, and this may be readily comprehended from the fact that they transmit the blood-vessels to the brain. The **cerebral sinuses** contain a small amount of blood and sometimes none.

Symptoms and Diagnosis.—In accordance with the mode of development and the course of the disorder a distinction is made between *acute*, *subacute*, and *chronic cerebral anemia.* The most acute form of cerebral anemia is probably that which develops in consequence of *vascular spasm.* Under such conditions there generally occurs a sense of oppression in the precordium, and often also palpitation of the heart. The face becomes pale; the patient manifests a tendency to yawn; nausea is present, and occasionally vomiting takes place. The extremities become heavy and the skin rough; ringing in the ears occurs, with spots before the eyes, and then obscuration of the field of vision and loss of the sense of hearing. The pupils become contracted, and finally the patient falls, with loss of consciousness, often with a slight cry, and general clonic muscular contractions. The duration of the loss of consciousness or fainting—syncope—varies from a few seconds to a few minutes. Restoration to consciousness occurs with varying rapidity.

Cerebral anemia due to hemorrhage gives rise to quite the same manifestations, except that these develop rather more slowly and in somewhat of a *subacute manner*. Chronic discharges are followed by the **clinical picture of chronic cerebral anemia,** consisting in part of irritative and in part of paralytic phenomena referable to the brain. The patients complain frequently of vertigo, headache, sleeplessness (agrypnia), ringing in the ears, spots before the eyes, and palpitation of the heart, and they not rarely exhibit delirium (of inanition). Often complaint is made of paresthesia in the lower extremities, and occasionally also of clonic muscular contractions and paresis. All of the symptoms are increased in the upright posture and may be followed by obscuration of the visual field, general clonic muscular spasm, loss of consciousness, and even death. The recognition of cerebral anemia is easy; the appearance of the patient and the etiology will suggest the *diagnosis.*

Prognosis.—The prognosis of cerebral anemia is favorable if the causative factors are curable and the condition of general anemia is not excessive.

Treatment.—In the presence of cerebral anemia it is always important to place the patient in the **horizontal posture,** with the **head dependent.** In other respects the treatment varies with the causative factors operative in the individual case—**causal therapy.** In the presence of vasomotor spasm due to fright stimulating remedies should be employed, as, for instance, application with friction to the forehead of cologne-water or vinegar, inhalation of ammonia, irritation of the nose with a feather, tickling or brushing the sole of the foot, subcutaneous injections of camphorated oil, from 5 to 10 drops of sulphuric ether internally, and the like. When the loss of blood has been large the extremities should be bandaged in order to force the blood into the internal viscera, and **transfusion or infusion** of physiologic salt-solution should be practised. When the amount of hemoglobin is deficient **preparations of iron** will be indicated, and when wasting discharges have taken place a **nutritious diet.**

CEREBRAL HYPEREMIA.

Etiology.—Extremely little of a definite nature is known with regard to the clinical significance of cerebral hyperemia. At any rate, but subordinate importance can as yet be attached to it. In the first place, a distinction must be made between *arterial* and *venous hyperemia of the brain*, accordingly as the condition is dependent upon an abnormal increase in the supply of arterial blood or upon interference with the escape of venous blood. Arterial hyperemia of the brain has also been designated congestive or active, and venous hyperemia, passive or hypostatic. *Arterial hyperemia of the brain* may result from **increased activity on the part of the heart-muscle**, such as occurs transiently following psychic and physical exertion, after a heavy meal and the use of large amounts of alcohol, and persistently in the presence of hypertrophy of the left ventricle of the heart from any cause (aortic valvular insufficiency, arteriosclerosis, contracted kidney, idiopathic hypertrophy of the heart). At times cerebral hyperemia develops because the **supply of arterial blood to certain portions of the body** is interfered with. Such a condition is observed in association with abdominal tumors, ascites, peritonitis, constipation, and aortic stenosis. Cerebral hyperemia may result also from **paralysis of the vasomotor nerve**, which arises from the cervical sympathetic. Thus, a number of poisons (alcohol, amyl nitrite, nitroglycerin) are capable of causing paralytic arterial hyperemia of the brain.

The most common variety of *venous hyperemia of the brain* depends upon **conditions of weakness involving the muscular wall of the right side of the heart**, which render impossible complete emptying of the right ventricle with the systole, so that blood-stasis extends from this cavity to the right auricle, thence to both venæ cavæ, and from the superior vena cava also to the internal jugular veins, the sinuses of the dura mater, and the cerebral veins. Such conditions develop with especial frequency in the presence of valvular lesions of the heart and diseases of the myocardium, the pericardium, and the respiratory organs (pulmonary emphysema, chronic bronchial catarrh, extensive pleural adhesions). At times venous hyperemia of the brain results from **pressure upon the superior vena cava** or **its peripheral branches**, such as may readily occur in the presence of an aortic aneurysm, a mediastinal tumor, an enlarged thyroid gland, and enlargement of the cervical lymphatic glands. **Thrombosis of the internal jugular veins** or of the cerebral sinuses should also be mentioned among the causes of hypostatic hyperemia of the brain. It should not be overlooked that **expulsive efforts** may give rise to blood-stasis. Therefore, those who engage in the act of blowing, who suffer from cough and from constipation, and who are engaged in arduous pursuits frequently suffer from venous hypostatic hyperemia of the brain. The condition finally develops in connection with **asphyxia.**

Anatomic Alterations.—Cerebral hyperemia is attended with **change in the color of the brain,** the white matter assuming a pale-rose color, while the gray matter acquires almost a brownish-red appearance. On transverse section the vessels appear distended with blood. Generally there are present also numerous capillary hemorrhages, which result from the rupture of small blood-vessels and extravasation of blood into the adventitial lymph-spaces and after rupture of the lymph-sheaths into the adjacent brain-tissue itself. The **pia mater** is conspicuous for the marked fulness and tortuosity of its veins. The sinuses of the dura mater generally contain considerable amounts of blood-clot and fluid blood. In the presence of chronic hyperemia of the brain the Pacchionian bodies appear highly vascular and markedly increased in size. The dura mater generally is densely distended, and presents bloody deposits upon its outer aspect after removal of the calvarium.

Symptoms and Diagnosis.—The symptoms of cerebral hyperemia, like those of cerebral anemia, consist in part of irritative and in part of paralytic phenomena referable to the brain. None of them, however, is distinctive of cerebral hyperemia, and therefore the diagnosis is, as a rule, based upon the presence of conditions capable of causing cerebral hyperemia, with the development of cerebral manifestations that do not appear to be dependent upon any other cerebral lesion.

In the presence of **arterial hyperemia of the brain** the patients usually complain of a sense of fulness in the head, of beating and pulsation in the skull, of ringing in the ears and impaired hearing, of spots before the eyes, vertigo, headache, anxiety, and palpitation of the heart. The face generally is greatly reddened. Often the patients suffer from sleeplessness. They are conscious of mental confusion, a sense of pressure in the head, and an indisposition or even an inability to engage in mental work. In some patients delirium occurs. There may be, further, loss of consciousness and general or local clonic muscular contractions. Paresthesiæ, paresis, and paralysis have also been observed in one or more extremities or upon one side of the body.

The symptoms of **venous hyperemia of the brain** do not differ materially from those of arterial hyperemia. The latter is attended with marked pulsation of the arteries, while the former is characterized by distention and tortuosity of the veins, and the arterial pulse is small. The face is more or less markedly cyanotic. In addition there will be the presence of some condition capable of causing stasis.

The *course of cerebral hyperemia* may be acute, subacute, or chronic, in accordance with the active causes. From the nature of many of the etiologic factors cerebral hyperemia is likely to *recur* from time to time.

Prognosis.—The prognosis depends essentially upon whether the causative factors are curable or incurable.

Treatment.—In the presence of any variety of cerebral hyperemia the **head** should be elevated; thereby the arterial circulation is rendered difficult and venous circulation is facilitated. Otherwise strict **causal therapy** should be enforced. In addition, an **ice-bag** should be applied to the head, and if constipation be present steps should be taken to secure a daily **evacuation of the bowels.** In the presence of arterial hyperemia of the brain **bleeding** may be advantageous.

CEREBRAL EDEMA.

Cerebral edema is difficult of recognition even anatomically. Upon section the brain appears moist and pale. Generally the ventricular and the cerebrospinal fluid is increased in amount. The arachnoid connective-tissue septa generally present edematous infiltration and swelling. The surface of the brain not rarely is conspicuously flattened. Cerebral edema probably is often an antemortem manifestation, and then is the result of circulatory disturbances. In addition to blood-stasis, it occurs also in connection with anemic and cachectic states, because under such circumstances the permeability of the blood-vessels is abnormally increased. Symptoms peculiar to cerebral edema are not known, and the condition can therefore not be recognized during life.

CEREBRAL HEMORRHAGE (ENCEPHALORRHAGIA).

Etiology.—Cerebral hemorrhage is one of the *commonest affections of the brain.* It results from antecedent disease of the small cerebral arteries, giving rise to the formation of **miliary aneurysms.** The latter exhibit a marked tendency to rupture, an occurrence that may take place spontaneously or be brought about by certain **contributory factors.** The disease of the blood-vessels is in part a *senile alteration*, and this explains the fact that in the majority of cases cerebral hemorrhage occurs after the fortieth year of life. *Presenile disease of the blood-vessels* of the brain occurs in connection with valvular disease of the heart, nephritis, in the sequence of alcoholism, lead-poisoning, syphilis, and other infectious diseases, and under such circumstances cerebral hemorrhage may occur even in young persons.

It must be admitted that obese, thick-set persons, with a short, thick neck, are more frequently seized with cerebral hemorrhage than spare and tall persons, so that there is a certain justification for the use of the designation *apoplectic habit.* The connection between both phenomena is, however, scarcely other than that disease of the blood-vessels occurs with especial frequency in obese persons. Nor can it be denied that cerebral hemorrhage occurs with particular frequency in certain families, so that an *hereditary predisposition* has been spoken of. Here also the heredity bears upon the tendency to disease of the blood-vessels.

Hemorrhage into the brain may occur during most profound rest, as, for instance, in sleep, but often its occurrence is favored by certain *contributory influences,* all of which are essentially operative by causing temporary increase of the pressure in the aortic system. Among these influences are anger and sudden emotional disturbance, hard work, powerful expulsive effort, the ingestion of a large meal, the use of alcohol, persistent stooping, and the like. Hypertrophy of the left ventricle, in consequence of nephritis, insufficiency of the aortic valve, or arteriosclerosis, also favors the occurrence of cerebral hemorrhage. *Atmospheric influences* appear possibly to have some etiologic effect, as cerebral hemorrhage not rarely increases in frequency with the onset of winter, perhaps because as a result of the influence of the cold the cutaneous blood-vessels contain less blood and the internal viscera receive an excess of blood.

Anatomic Alterations.—Two varieties of cerebral hemorrhage occur, namely, the *punctate* or *capillary* and the *massive* or *focal. Capillary hemorrhage* into the brain is without clinical significance. The extravasations are at most as large as the head of a pin, and they are distinguished from transverse sections of the blood-vessels by the fact that they cannot be brushed away with the finger. They result from rupture of the walls of small cerebral vessels, with the escape of blood into the adventitial lymph-space, and the development of aneurysmal dilatations in places. Rupture of the lymph-sheath then also occurs frequently, and the blood is forced into the adjacent cerebral tissue without causing material injury. Only when punctate hemorrhages into the brain are present in large number in close proximity with one another may they acquire the clinical significance of a massive hemorrhage, and give rise to focal symptoms. Capillary hemorrhages are especially frequent and numerous in the presence of cerebral hyperemia, infectious diseases, diseases of the blood, and after general clonic muscular convulsions.

That which follows refers only to *massive hemorrhage into the brain.* Such hemorrhage may take place into any part of the brain, although experience has shown that in the large majority of cases it occurs from a definite branch of the middle cerebral artery or the artery of the Sylvian fossa. At the base of the brain this vessel sends branches to the large basal ganglia, and among them rupture takes place with especial frequency in those arteries that supply the outer portion of the lenticular nucleus with blood. From this point the blood finds its way into the adjacent internal capsule, the optic thalamus, and the centrum semi-ovale. For the development of the most common clinical manifestations of cerebral hemorrhage (cerebral paraplegia upon the opposite side of the body) involvement of the middle third of the posterior limb of the internal capsule is essential, and through which the motor pyramidal tracts pass from the cerebral cortex and the corona radiata to the lower portions of the central nervous system. Accordingly, destruction of the capsule and hemiplegia occur only through indirect influences.

The *amount of blood extravasated* is susceptible of great variation. Extensive masses of blood often become apparent after removal of the calvarium through the greater tension of the dura mater upon the side of the hemorrhage than over the healthy hemisphere. When the dura is incised and is reflected it will often be conspicuous on account of its dry appearance over the affected hemisphere. The cerebral convolutions upon the affected side are flattened and expanded, while the intervening fissures are obliterated, obviously because the brain has been compressed from within against the inner surface of the skull. If the usual incisions through the body of the brain are made, the blood-clot comes into view. Often the blood has been forced through the basal ganglia of the brain and the internal capsule into the adjacent ventricle, and it may even pass from here through the foramen of Monro into the opposite lateral ventricle. It may also happen that the blood ruptures into the third ventricle, and from here partly escapes through the aqueduct of Sylvius into the fourth ventricle. Rupture outward through the cerebral cortex occurs far less commonly. The blood may be enclosed within a flattened extensive blood-clot, and possibly a portion of the blood may have entered the subarachnoid space of the spinal cord.

In recent cases the extravasation of blood represents a soft mass consisting of coagulated blood and lacerated brain-tissue. If the masses of blood are removed and rinsed in water, blood-vessels not rarely remain, upon which, often with the unaided eye, but better on microscopic examination, bulbous enlargements can often be seen—the miliary aneurysms. The space containing the extravasation of blood exhibits shreddy walls, which are frequently edematous and stained slightly yellow—so-called lemon-colored edema. Gradually macroscopic and microscopic changes take place in the extravasation of blood. The mass of blood becomes firmer, undergoes reduction in size, and acquires a brownish and subsequently rather a yellowish color. These changes depend upon absorptive processes and metamorphosis of the escaped red blood-corpuscles, which gradually undergo disintegration. Eventually the extravasation of blood consists almost exclusively of fatty granular cells and granular, flaky blood-pigment in the form of needles or plates, together with remains of vessels in a state of fatty degeneration. Should complete absorption of the extravasation occur, a clear or slightly turbid, rarely purulent fluid takes its place. In consequence of interstitial encephalitis a smooth connective-tissue capsule forms about the fluid, and in this way there results an **apoplectic cyst**, which persists for all time. Only a small hemorrhage will leave behind an **apoplectic cicatrix.** The blood is slowly absorbed, and in its place there develops a linear cicatrix constituted of neuroglia and stained deeply brown by hemoglobin.

Destruction of the motor pyramidal tract in the internal capsule is followed by **secondary degeneration** of this tract to the spinal aspect of the lesion, and accordingly atrophy of nerve-fibers and hyperplasia of neuroglia can be traced through the central portion of the foot of the related cerebral peduncle, the pons, the pyramidal decussation, the anterior pyramidal tract of the spinal cord upon the same side, and the lateral pyramidal tract upon the opposite side (Vol. I., p. 597, Fig. 83). In fresh specimens the alterations naturally can often not be recognized, while they at once appear distinctly in sections hardened in Müller's fluid.

Cerebral hemorrhage has a marked tendency to *recur*, inasmuch as a tendency to the formation of aneurysms is present. It is, therefore, not uncommon to find *several extravasations of blood* in the same brain, and which, as a rule, are of varying age, but often symmetric in distribution.

Symptoms.—In typical cases of cerebral hemorrhage *several stages* may be distinguished, and these are designated prodromes, apoplectic seizure, reactive manifestations, symptoms of deficiency, and secondary contractures. Nevertheless, it not rarely happens that some stages are wanting or are not reached in the individual case.

The stage of *prodromes* or of *premonitory symptoms* is characterized by attacks of fulness in the head, vertigo, beating in the head, excitability, impairment of memory, at times also by transient aphasia. Some patients complain for hours or days of unilateral formication or impaired cutaneous sensibility, or these manifestations appear in only a single member. Conditions of transient weakness or paralysis also occur in one or other member or in unilateral distribution—phenomena that are generally dependent upon small cerebral hemorrhages.

The occurrence of a considerable extravasation of blood into the brain gives rise to the so-called *stroke* or *apoplectic attack*. Not rarely the patient falls unconscious, like an animal struck by an axe. The loss of consciousness persists for hours or days, and often is so profound that the patient does not respond to vigorous cutaneous irritation, as, for instance, pinching of the skin. The pupils are not rarely unequal, and contract but sluggishly on exposure to light. Respiration often is snoring, at times irregular or of Cheyne-Stokes character, and the lips, particularly on the paralyzed side, flap to and fro with the respiratory movements like a curtain in the wind. The face is not rarely deeply reddened and congested, but at other times the patient presents a pallid and dejected appearance. The pulse is generally in accord with the appearance of the patient, being, in the one instance, tense and full, and in the other small and readily compressible. The pulse, further, may be slow and irregular. Absence of almost all of the cutaneous reflexes upon the paralyzed side of the body—as, for instance, the cremasteric, the abdominal, the gluteal, and the scapular—is of diagnostic importance. I have, however, often observed the mammillary reflex to be present, although it appeared more sluggish than upon the healthy side of the body. When unconsciousness is profound, **involuntary discharges** frequently occur, and especially the urine is often voided in bed. During the first twenty-four hours the urine is often excreted in increased amount (polyuria), and at times it contains albumin and sugar (remote influence upon the medulla oblongata).

In some patients repeated *vomiting* occurs; in others, unilateral or general *clonic convulsions*. The latter is the case especially when the extravasation has ruptured into the ventricles or toward the cortex. *Muscular contractures* also indicate involvement of the cortex. Occasionally *forced attitudes* are observed: the head and the eyes are turned in the same direction, and always return to the same position. I have often observed convulsive *trembling* of the entire body.

At times *abortive apoplectic attacks* occur (transitory vertigo, brief syncope, severe vomiting). It may also happen that an apoplectic attack is preceded by unilateral paralysis, and this is to be explained by the fact that an originally small extravasation of blood first injures the motor path, and only subsequently increases to such a degree as to give rise to the apoplectic seizure.

Several conditions are responsible for the *occurrence of an apoplectic attack.* In the first place, the sudden extravasation of blood and the resulting sudden mechanical shock to the brain, and also the rapid increase in cerebral pressure, must be taken into consideration.

Restoration of consciousness after an apoplectic attack takes place in various ways, and occurs at times rather abruptly and at other times quite gradually. Often the patient is unconscious of the last events that took place before the attack. In severe cases consciousness does not return at all, and death occurs in profound unconsciousness from cardiac or respiratory paralysis in consequence of excessive cerebral pressure.

The *stage of reaction* is characterized by great restlessness on the part of the patient, with elevation of temperature. The patient frequently throws himself restlessly about in bed, becomes delirious, and talks incessantly, while the temperature may reach as high as 39° C. (102.2° F.), rarely higher. These symptoms generally appear in the course of from three to five days, and they usually persist for only a few days.

The **paralytic symptoms** or the **symptoms of deficiency** vary in accordance with the seat of the extravasation of blood, and are governed entirely by the diagnostic rules already given. When destruction of the internal capsule has taken place ordinary cerebral hemiplegia occurs upon the opposite side of the body. Under such conditions the facial and the hypoglossal nerve and the arm and the leg are paralyzed. It is noteworthy that in the distribution of the facial nerve the frontal muscle, the corrugator of the brow, and the orbicular muscle of the eyelids remain intact, and that, besides, the arm is frequently more completely paralyzed than the leg. Sensory disturbances generally are wholly wanting, while at other times, in addition to hemiplegia, there is transitory hemianesthesia. Rarely, persistent hemianesthesia occurs, and this is indicative of destruction of the posterior third of the posterior limb of the internal capsule.

On careful examination one can readily convince himself that also the *muscles of the trunk* upon one side are involved in the paralysis. The shoulder is lower (paralysis of the trapezius), the thorax participates less in respiration, and the expulsive power of the abdominal muscles is enfeebled. I have often found impaired mobility of the corresponding true *vocal band.* At times symptoms of *sympathetic paralysis* occur (unilateral redness of the face, unilateral sweating, retraction of the eyeball, narrowing of the palpebral fissure, contraction of the pupil).

In the further course of the disease a portion of the paralytic phenomena subside, at times rapidly, at other times more slowly. Improvement in the leg occurs earliest and in greatest degree, the peronei alone generally remaining paralyzed, and for this reason the patient in walking first applies the anterior portion and the outer border of the foot upon the ground, and often scrapes the toes. Less improvement takes place in the arm. The involvement of the extensors upon the dorsal aspect of the forearm remains particularly severe. Long-continued paralysis gives rise to **atrophy of the affected muscles from disuse.** The **electric irritability** remains unaltered in cases of cerebral paralysis.

The occurrence of **associated movements** is noteworthy. In laughing, crying, and sneezing the paralyzed muscles of the face are distorted. Cutaneous irritation, for instance, such as may be excited by a fly, induces involuntary elevation of the paralyzed arm. Strong pressure with the healthy hand may excite a similar movement in the paralyzed hand. *Symptoms of secondary contractures* develop progressively, and these have without adequate reason been attributed to the secondary degeneration of the pyramidal tracts in the spinal cord. Movements at the ankle-joint can be executed only with difficulty. The fingers are flexed, and can be extended only with an effort. The forearm is generally held in a flexed and pronated and but rarely in an extended position, and the upper arm is generally held in a position of adduction. In addition, the patellar, the triceps, and the periosteal reflexes are greatly exaggerated.

Every patient with cerebral hemorrhage is threatened with the risk of *recurrence.* The period of time within which this may occur and the frequency are beyond estimation in advance. Another danger consists in the greater or lesser degree of *dementia.* The patients become lacrimose and exhibit great and abrupt variability in mood. Their memory and their mental capacity in general become more and more greatly impaired. Often incontinence of the bladder and the rectum develops; eventually death results amid symptoms of profound exhaustion.

Diagnosis.—Cerebral hemorrhage may be readily confounded with other conditions of profound coma at the time of the apoplectic attack, as, for instance, with **alcoholic intoxication or opium-poisoning.** In the differential diagnosis consideration should be given to the history. In addition, the odor of the expired air should be investigated for the presence of alcohol; and if opium-poisoning be suspected, extreme contraction of the pupils should be looked for. **Uremic, diabetic,** and **septic coma** may be confounded with cerebral hemorrhage. The differentiation then depends especially upon the demonstration of albumin and tube-casts or of sugar in the urine, or of other symptoms of septicemia. At times **purulent** or **tuberculous meningitis** sets in with sudden loss of consciousness and hemiplegia. The presence of rigidity of the neck and of fever is indicative of meningitis. Attacks of loss of consciousness and paralysis occur also in connection with **tabes dorsalis, multiple sclerosis,** and **epilepsy,** but they are generally transitory. Similar conditions occur also in cases of **progressive paralysis of the insane,** in which, however, the profound mental disturbance is noteworthy.

Difficulty may further arise during the apoplectic attack in determining the side that is paralyzed, and therefrom the *seat of the hemorrhage.* Should the coma be not excessive, only the unaffected parts will be withdrawn and engage in movements of resistance on pinching and pricking the skin, while the paralyzed parts remain unmoved. It will be noted further that the arm upon the healthy side when raised remains elevated, or falls but slowly, whereas that upon the paralyzed side falls like an inanimate body. In the presence of profound coma the tests mentioned are untrustworthy. Under such circumstances the *state of the cutaneous reflexes* should be studied, particularly the cremasteric and the abdominal, which are absent upon the paralyzed side. The focal symptoms will indicate the *seat of the hemorrhage* into the brain. With regard to the *differential diagnosis between cerebral hemorrhage and thrombosis or embolism of the cerebral arteries,* which, by the way, can never be made with certainty, the succeeding section should be consulted (p. 62).

In rare cases all of the symptoms of cerebral hemorrhage appear, although it is impossible to demonstrate anatomic changes in the brain.

Prognosis.—The prognosis of cerebral hemorrhage is grave. Even after the patient has emerged from the profound unconsciousness attending an apoplectic attack, and after the paralysis has in part subsided, sudden aggravation may take place and lead to a fatal termination within a short time. Often, though not invariably, such a condition is dependent upon renewed hemorrhage. Experience has shown that progressive elevation of temperature at the time of the apoplectic seizure and a greater degree of paralysis in the leg than in the arm are unfavorable phenomena.

Treatment.—The treatment of cerebral hemorrhage varies in accordance with the several stages of the disorder. At the time of the apoplectic attack the **head should be elevated** and **venesection** practised if the patient presents a congested appearance and a tense pulse, 500 c.c. of blood being withdrawn. At times patients emerge from coma immediately at the conclusion of the bleeding. In cases in which the face is pale and the pulse small stimulants should be employed, as, for instance, spirituous applications to the temporal regions, inhalation of ammonia, mustard-plasters over the heart and to the calves of the legs, subcutaneous injections of camphorated oil, and the like. At the time of the apoplectic attack an **ice-bag** may in addition be applied over the side of the head on which the hemorrhage is suspected to have taken place, in order, through the stimulating influence of the cold, to cause contraction of the blood-vessels and closure of the opening. Attempts to administer food should be made only after consciousness has been restored and the power of swallowing is present.

In the stage of *reactive phenomena* the *ice-bag* should be continued upon the head. The **food** should be bland and liquid, and consist preferably of milk. Coffee, tea, and alcohol should be avoided. A **daily evacuation of the bowels** should be secured by the administration of stewed apples or prunes, or by means of rectal injections of water, or a glycerin-enema, or a teaspoonful of compound licorice-powder, or compound infusion of senna (compound infusion of senna, 180.0—6 fluidounces; sodium sulphate, 20.0—5 drams: 15 c.c.—1 tablespoonful—thrice daily). In the treatment of the *paralytic symptoms* or *symptoms of deficiency* especial attention should be given to the **regulation of the diet**, to the **nursing**, and to **massage** of the paralyzed members.

In the *diet* all articles of food difficult of digestion and of an irritating character should be avoided. In the case of bedridden patients care should be taken that the *sheets* are smooth, and all foreign bodies should be removed, particularly dry bread-crumbs, in order to avoid bed-sores. It is advisable to cleanse the skin over the sacrum and the buttocks twice daily with a solution of mercuric chlorid (1.0 : 1000) by means of cotton, and then to rub upon the surface cologne-water, vinegar, or alcohol. Massage should be applied once daily to the paralyzed members. It is important to subject the joints to passive movement. In many instances I have, by means of massage, either wholly prevented the occurrence of secondary muscular contractures or restricted them within narrow limits.

Sorbefacients, especially potassium iodid, are largely employed; but it is extremely doubtful whether they really exert any sorbefacient effect. Likewise, nothing is to be expected from the catalytic and consequently sorbefacient effect of a *galvanic current* applied to the skull. In the treatment of the paralysis all sorts of *nervines* have been recommended, among others *subcutaneous injections of strychnin*, although these appear to have yielded no definite results. The results from properly practised massage also are better than those from *faradization* or *galvanization* of the paralyzed parts. Differences of opinion exist as to the value of *courses of treatment at baths*. Saline baths and indifferent thermal baths (Ragaz, Pfäffers, Wildbad, Gastein) have been variously recommended.

For the relief of the *secondary contractures* **massage** and **Swedish movements** may be employed; the **galvanic current** also favors the correction of contractures.

THROMBOSIS AND EMBOLISM OF THE CEREBRAL ARTERIES.

Anatomic Alterations.—If a blood-vessel of the brain is occluded by a foreign body, the part of the brain deprived of its blood-supply dies and undergoes **necrotic softening—necrotic encephalomalacia.** Often such necrosis is associated with extravasation of blood, and the dead tissue acquires the appearance of red softening, which subsequently becomes transformed into yellow, and then into gray softening, accordingly as the hemoglobin of the emigrated red blood-corpuscles undergoes disintegration in progressive degree, becomes transformed into hematoidin (resembling biliary coloring-matter), and undergoes absorption. Should extravasation of blood not occur, the dead tissue appears white or gray, and is conspicuous especially on account of its soft, at times almost diffluent, consistency. Microscopic examination discloses in the softened tissues especially fatty granular cells; besides red blood-corpuscles, hemoglobin, and disintegrating nerve-fibers and ganglion-cells in a state of fatty degeneration. A few ganglion-cells not rarely present calcification. Should absorption of the softened brain-tissue take place, a **cyst** results that completely resembles a hemorrhagic cyst. At times **multiple foci of softening** occur in the brain, for embolism, as well as thrombosis, exhibits a tendency to recur.

The changes that take place in an area of necrotic softening of the brain resemble entirely those of hemorrhagic and inflammatory softening. The presence of arterial occlusion is alone distinctive of the first of these conditions. In cases of long standing, however, the arterial occlusion may have undergone absorption, and it will then be difficult or impossible to distinguish between hemorrhagic, inflammatory, and necrotic softening of the brain.

If fibrinous deposits have taken place in a cerebral vessel itself, the process is designated *thrombosis*, while if, on the other hand, foreign bodies are swept with the blood-current from the heart into the cerebral arteries, the condition is one of *embolism*. The differentiation between the two conditions is not always easy. For the diagnosis of embolism the demonstration of a source for an embolus is necessary, most frequently valvular disease of the heart. In addition, emboli are frequently found riding at the point of division of an artery, so that their extremities extend into the two branches of the vessel. Thrombotic deposits occur especially at such points in the vessels as calcification, fatty degeneration, or endarteritic thickening has taken place.

Necrotic softening of the brain will fail to take place after occlusion of an artery only when the vessel to the peripheral aspect of the point of occlusion communicates with the branches of adjacent arteries, and thereby rapid establishment of a collateral circulation is possible. In this connection the conditions in the arteries of the cerebral cortex are more favorable than those in the basal ganglia. For the occurrence of embolic softening of the brain it is additionally necessary that the obstructing plug should be free from bacteria capable of exciting inflammation, as otherwise inflammation of the brain—encephalitis—would result.

Etiology.—The most common source for cerebral embolism is **valvular disease of the heart** at either the aortic or the mitral orifice, if thrombotic vegetations become detached from the valves. Less commonly emboli are derived from thrombotic deposits upon **arteriosclerotic areas in the aorta** or from **aneurysm of the aorta. Cardiac thrombi, tumors,** and **echinococcus-cysts of the myocardium,** as well as **thrombosis of the carotid artery,** also may give rise to cerebral embolism. Rarely, cerebral embolism is associated with **disease of the lungs.** Nevertheless, cases of pulmonary abscess, carcinoma and gangrene, and of putrid bronchitis are known in which embolic metastasis of detached portions of lung-tissue to the brain has taken place. **Thrombi in the pulmonary veins** may disintegrate and be carried into the brain.

So-called *crossed cerebral embolism* is worthy of note, although it occurs but seldom. The condition is possible only when the *foramen ovale* has remained patulous. If under such conditions thrombosis has developed in a peripheral vein of the body, it is possible that detached portions thereof may gain entrance from a vena cava into the right auricle, and hence through the patulous oval foramen into the left auricle, then to be swept with the blood-current into the left ventricle, the aorta, and eventually into a cerebral artery. A similar occurrence may take place in connection with neoplasms that have perforated into peripheral veins and have undergone disintegration.

Thrombosis of a cerebral artery may, in the first place, be the result of arteriosclerotic or fatty changes in the cerebral vessels, such as occur especially at advanced age and after severe disease (marantic thrombosis). Arteriosclerotic changes may occur at a presenile period as a result of syphilis, nephritis, alcoholism, and lead-poisoning. *Compression-thrombosis*, resulting from constriction of a vessel by pressure, occurs but seldom, and likewise *inflammatory thrombosis* and *thrombosis by extension*, the latter from thrombosis of the carotid or vertebral artery extending into the cerebral artery.

Embolism and thrombosis occur more commonly in some cerebral arteries than in others. The *left middle cerebral artery* or *artery of the Sylvian fossa* is the seat of embolism with especial frequency, and this preference is explained by the fact that the left carotid viewed from the commencement of the aorta is more directly a continuation of the aorta, while the right innominate, and with it the right carotid, arises from the arch of the aorta almost at a right angle. Naturally, a foreign body in the blood will more readily enter the former than the latter. Cerebral thrombosis develops with especial frequency also in the left middle cerebral artery. Next in frequency the basilar artery is involved.

Symptoms and Diagnosis.—If the main trunk of the left middle cerebral artery is occluded by embolism or thrombosis, the inferior frontal convolution, the anterior and posterior central convolutions, and the superior temporal convolution undergo necrosis, and **right-sided cerebral hemiplegia and aphasia** are a necessary consequence (p. 42, Fig. 13). Occlusion of the basilar artery is attended with the pontine symptoms described on pp. 27 and 28. Obstruction of those branches of the Sylvian artery that supply the basal ganglia is attended with involvement of the posterior limb of the internal capsule, and gives rise to **ordinary cerebral hemiplegia.** The cerebral symptoms in themselves are in nowise distinctive of embolism or thrombosis. It frequently happens also that the onset of the disorder, like that of cerebral hemorrhage, is sudden and attended with symptoms of an **apoplectic attack,** and that also the further course of the disorder corresponds exactly with that of cerebral hemorrhage. From what has been said it must be evident that the differentiation between cerebral hemorrhage and embolic or thrombotic softening of the brain is extremely difficult, if at all possible. Even a youthful age, the demonstration of a valvular lesion of the heart, right-sided hemiplegia, and aphasia are not sufficient to justify a positive conclusion that embolism is present; and also the statements that frequently the apoplectic attack is wanting, especially in connection with thrombosis, that the disorder not rarely sets in with clonic muscular spasm, that at the time of the apoplectic attack the face is pallid and the temperature of the body does not fall, and the like, are untrustworthy indications.

The **prognosis** is serious, and corresponds with the prognosis of cerebral hemorrhage as indicated on p. 59.

Treatment.—The treatment is the same as that for cerebral hemorrhage, except that at the time of the apoplectic seizure **stimulants** should be employed instead of bleeding.

INFLAMMATION OF THE BRAIN (ENCEPHALITIS).

Inflammation of the brain—encephalitis—occurs in various anatomic and clinical forms. *Cerebral abscess* is the condition best known, while our knowledge of *hemorrhagic encephalitis*, *infantile polio-encephalitis*, and *congenital encephalitis* is much more scanty.

CEREBRAL ABSCESS.

Etiology.—Cerebral abscess probably occurs scarcely other than as a result of the entrance of pyogenic **bacteria** into the brain. Several bacteria may play such a *role*, especially the Streptococcus and the Staphylococcus pyogenes, the pneumoniacoccus, and the influenza-bacillus. In isolated cases the ray-fungus (actinomyces) and the thrush-fungus (Oïdium albicans) have also been found in the blood as the cause of suppuration. If suppurating solitary tubercles in the brain are considered as true cerebral abscesses, tubercle-bacilli, which, it is true, are often associated with pyogenic cocci, will also have to be included among the excitants of cerebral abscess. Further, the demonstration of bacteria can be anticipated only in recent cerebral abscesses, as the older abscesses become gradually sterile, the contained bacteria dying off. Either bacteria gain entrance into the brain with emboli, or they penetrate from the neighborhood—*cerebral abscess by extension*; or, finally, a channel of communication with the brain from without is established directly through traumatism—*traumatic abscess of the brain*. Cerebral abscess occurs most commonly as a result of extension from adjacent disease, and particularly from disease of the internal ear (otogenic abscess of the brain).

Embolic abscess of the brain may result from **ulcerative endocarditis.** Under such conditions bacterial plugs become detached from the inflamed endocardium, and gain entrance into the brain with the arterial blood-current. **Purulent and putrid destructive processes in the air-passages or in the pleura** also occasionally constitute the source for an embolic abscess of the brain, as, for instance, pulmonary abscess and gangrene, putrid bronchitis, bronchiectasis, and even pleural empyema.

At times abscess of the brain develops in the sequence of **infectious diseases**, as, for instance, influenza, septicopyemia, erysipelas, and probably also under other circumstances the bacteria gain entrance into the brain generally with the blood-stream.

In cases of *traumatic abscess of the brain* (open fracture of the skull, gunshot and contused wounds) bacteria may gain entrance into the brain with foreign bodies or immediately from the air.

Further, cerebral abscess has occasionally been observed to develop after violent concussion of the skull, obviously because the concussion has lowered the resistance of the brain-tissue and thereby rendered it more susceptible to the influence of bacteria, which frequently circulate in the blood even in health.

Among *abscesses of the brain resulting by extension from adjacent disease* those that develop in consequence of **inflammation of the internal ear** occur with especial frequency, and in this connection not alone acute, but also chronic inflammations that have temporarily assumed an acute character are operative. Cerebral abscess is occasionally associated also with inflammation of the frontal sinuses, the nose, the nasopharynx, the antrum of Highmore, and the orbit, the cranial bones, the cerebral sinuses and meninges, erysipelas of the head, parotiditis, and inflammation of the connective tissue of the neck.

Abscess of the brain may occur at any time of life.

Anatomic Alterations.—A distinction must be made between *encapsulated* and *free abscess of the brain*, accordingly as the collection of pus is surrounded by a connective-tissue membrane or not. Obviously, an encapsulated abscess of the brain must result from a free collection of pus, and it is therefore not surprising that at times such collections are found in the brain that have undergone only partial encapsulation. It can be readily understood that a free abscess of the brain is of far more serious significance than an encapsulated abscess, as but slight resistance is offered to its extension and its rupture into the ventricle or through the pia into the intermeningeal space.

The *size of a cerebral abscess* is susceptible of great variation. At times the collection of pus may be barely visible, while at other times it may involve the larger part of a cerebral hemisphere. Abscesses of the brain may be *single* or *multiple*. The latter result especially from septicopyemic causes. The pus from a cerebral abscess in many cases resembles greenish cheesy abscess-pus, and is found on microscopic examination to contain, in addition to (usually polynuclear) round cells, also fatty granular cells, crystals of fatty acids, and plates of cholesterin. At times the pus has acquired, a dense, viscid, and gelatinous consistency. The pus, further, may have an offensive odor if the pyogenic cocci are derived from putrid foci (pulmonary gangrene, putrid bronchitis, otitis, inflammation of a sinus). Although collections of pus may take place in any part of the brain, they occur *most commonly* in the *temporal lobe* and the *cerebellum*, because otogenic abscesses of the brain are generally located in these two situations.

Symptoms and Diagnosis.—Even extensive abscesses of the brain may be wholly unattended with symptoms, and be discovered accidentally on postmortem examination—*latent abscess of the brain.* In some cases cerebral abscess presents the clinical picture of progressive **exhaustion** or of **septicopyemia,** of an **apoplectic attack,** or of a suddenly developed **psychopathy.** In other instances the **general** or the **local cerebral manifestations** direct attention to the brain, and especially in view of the causative factors will a conclusion be reached as to the presence of a cerebral abscess. Specific symptoms of cerebral abscess are not known. Nevertheless, elevation of temperature and chills are noteworthy symptoms, although cases of cerebral abscess occur in which the temperature pursues a subnormal course. Among the *general cerebral symptoms* headache, vertigo, staggering gait, vomiting, inequality of the pupils, and irregularity and slowing of the pulse occur with especial frequency. In addition, coma and at times choked disc should be mentioned. On palpation the head will be found tender in places, although not much information of value in the localization of a cerebral abscess is to be obtained in this way.

At times **meningitic symptoms** (rigidity of the neck, delirium) preponderate, so that there may be danger of assuming the existence of cerebrospinal meningitis and overlooking the presence of a cerebral abscess. Naturally, secondary purulent meningitis may be readily superadded to cerebral abscess, and such cases almost always pursue an unfavorable course. Sometimes **clonic muscular spasm** occurs in one or another extremity, upon one side or generally. Should the spasms always be repeated in the same member, or if they always begin in the same member and extend to the remaining members in a manner corresponding to the arrangement of the motor cortical centers, this will be evidence that the cortex is involved by the abscess. Aphasic disturbances occur not rarely in cases of cerebral abscess, as the left temporal convolutions are frequently the seat of the purulent collection. Persons with cerebral abscess are exposed to the greatest danger, as they may be destroyed by septicemia or central paralysis, or to the cerebral suppuration is superadded a generally fatal purulent meningitis, or the pus may rupture into the ventricles or externally through the cortex and induce a fatal issue amid general clonic muscular spasm. In the presence of an open wound of the skull it may happen that brain-tissue admixed with pus escapes externally.

Prognosis.—In accordance with the statements just made the prognosis of cerebral abscess is quite grave. Relief can be expected only from surgical intervention. Multiple and unencapsulated abscesses are especially unfavorable, for success can scarcely be obtained from surgical measures in the presence of the former, as it would be impossible to discover and incise all of the abscesses; while the latter have a tendency to extend and to rupture.

Treatment.—**Prophylactic measures** are applicable especially in the presence of disease of the internal ear. It is particularly important to treat chronic inflammation of the middle ear with the utmost care. Nothing can be accomplished in the treatment of abscess of the brain by means of internal remedies. The single mode of relief consists in **operative incision and evacuation of the abscess,** and this will naturally require a diagnosis of the situation of the pus. If after opening the skull no abscess is visible upon the surface of the brain, deep puncture should be made into the structure of the brain, and if pus appears an incision into the brain should follow.

HEMORRHAGIC ENCEPHALITIS.

In the presence of hemorrhagic encephalitis the diseased portion of brain is characterized by excessive softness and a red, blood-suffused color; a greater or smaller number of minute extravasations of blood are found at the periphery. The condition is therefore designated also *inflammatory encephalomalacia*, and it may readily be confounded with hemorrhagic, embolic, and thrombotic softening. The area of softening may, like the conditions last named, pass from red to yellow, gray, and white stages. If the area of softening, which on microscopic examination will be found to consist of fatty granular cells, degenerated nerve-fibers, and ganglion-cells in a state of fatty degeneration, and blood-vessels involved in fatty degeneration, is absorbed, there remains an *inflammatory cyst*, and under some conditions an *inflammatory cicatrix*. Often several areas of inflammatory softening occur in the brain—*multiple encephalitis*. The size of the individual foci is susceptible of great variation. That it is often exceedingly difficult, and even impossible, to differentiate inflammatory softening of the brain from hemorrhagic, embolic, or thrombotic softening has been pointed out in preceding sections. The distinction depends especially upon the demonstration of emboli or thrombi in a cerebral artery, or upon the clinical course.

Etiology.—The *causes* of hemorrhagic encephalitis must occasionally be looked for in antecedent **infectious disease**; the disorder has often been observed to develop after influenza. Next in frequency **traumatism** and **alcoholism** may be mentioned as causes. In two cases under my observation the disease depended upon intergyral *chronic meningitis*.

Symptoms.—The disease frequently sets in with severe **headache** and with repeated **vomiting**. **Elevation of temperature** takes place and **consciousness** becomes more and more impaired. Occasionally **rigidity of the neck, inequality of the pupils,** and **irregularity of the pulse** are observed. Often **spasm** and **paralysis** occur in one extremity, and the latter may extend progressively and be gradually transformed into hemiplegia, because the inflammation extends upon the cortex from one motor center to the next adjacent. As a rule, the disease pursues too rapid a course to permit of the development of papillitis. Death results from central paralysis in the presence of progressive loss of consciousness. Recovery occurs but rarely.

Treatment.—Attempts should be made to control the inflammation by the application of an **ice-bag** to the head, and in the presence of severe headache **antipyrin** or **phenacetin** will be prescribed internally. Should this not suffice, a subcutaneous injection of morphin should be given.

ACUTE CEREBRAL PARALYSIS OF CHILDHOOD (ACUTE INFANTILE POLIOENCEPHALITIS).

Etiology.—The disease is suggestive in its clinical course, and probably also in its anatomic alterations, of acute spinal paralysis of childhood. As a rule, it attacks children *between the first and the fourth year of life.* At times it develops in the sequence of *infectious diseases*, as, for instance, measles, scarlet fever, pharyngeal diphtheria, parotiditis, whooping-cough. In other instances it occurs as an *independent disorder*, as has been assumed as a result of primary infection of the motor cortical centers.

Symptoms.—Often the disorder sets in suddenly with a **chill, high fever, loss of consciousness,** and **clonic muscular spasm,** while in other cases headache has preceded as a *prodrome.* The muscular spasm may be confined to one extremity, or involve one-half of the body, or it may be general. After one or two days the patient generally emerges from his unconsciousness, but now it is observed that **paralysis** has developed. This involves at times but a single extremity (monoplegia), although it may occur in the form of hemiplegia and rarely as paraplegia. Although cerebral nerves may participate in the paralysis, the facial nerve not rarely remains uninvolved, in spite of the existence of hemiplegia. Sensory disturbances are generally wanting. The electric irritability of the paralyzed nerves and muscles exhibits no alteration. After a time the paralyzed muscles undergo gradual wasting. The affected members remain backward in growth and appear shortened. Muscular contractures develop, and the tendon-reflexes, particularly the patellar, the Achilles, and the triceps, and also the periosteal reflexes, are increased. The disease has therefore been designated spastic infantile paralysis. Should right-sided paralysis occur in a child already able to speak, aphasia may develop. Often the children remain idiotic or suffer from epilepsy throughout life. At times choreoid or athetoid movements or associated movements occur in the paralyzed members. Occasionally the paralysis subsides in part, rarely in full—temporary paralysis.

Diagnosis.—Infantile polioencephalitis is distinguished from other varieties of spastic paralysis of childhood, such as follow obstruction to birth and cerebral hemorrhage and embolism, by its febrile onset.

Anatomic Alterations.—It has been thought that the disease is analogous to acute spinal paralysis of childhood, and that it depends upon acute hemorrhagic inflammation of the motor cortical centers in the central convolutions, but convincing pathologic evidence is yet wanting. In cases in which post-mortem examination has been made many years after the onset of the disorder defects, often funnel-shaped, have been found in the brain —*porencephaly*—or cerebral sclerosis. Secondary degeneration takes place in the pyramidal tracts.

The **prognosis** is grave, as death may take place during the febrile attack, and, besides, the clinician is powerless in the presence of the disease.

Treatment.—The best results are obtained from **massage** and **orthopedic treatment** of the paralyzed members, by **baths** and **mental training.**

CONGENITAL ENCEPHALITIS.

Congenital encephalitis is as yet only of anatomic interest. The condition is characterized by accumulations of fatty granular cells in the brain of newborn children whose mothers, frequently, have died from *infectious disease* (small-pox, syphilis). Further, it has not even been demonstrated that the collections of fatty granular cells result in all cases from inflammatory processes, and are not dependent upon natural developmental phenomena in the brain.

CEREBRAL NEOPLASMS (TUMORS OF THE BRAIN).

Etiology.—Tumors of the brain are at times dependent upon **traumatism** or **infectious disease.** I have observed glioma of the brain develop in one case after a fall upon the occiput and in another after a blow upon the head with a ruler. Tuberculosis and syphilis at times give rise to the development of solitary tubercles or gummata in the brain. At times tumors of the brain develop by **metastasis.** Thus, I have observed secondary carcinoma of the brain develop secondarily to carcinoma of the esophagus. Neoplasms may originate also by extension from adjacent disease. Meningeal tumors particularly at times press into the brain-tissue and thereby acquire the importance of a brain-tumor. Often, however, no cause can be elicited for the tumor in the brain. Cerebral neoplasms may occur at *any time of life*, and they appear more commonly in *males* than in females, although they are among the *less common disorders.*

Anatomic Alterations.—Cerebral tumors are not confined to any definite situation, and are at times seated on the surface and at other times in the depth of the brain. Most commonly but a single neoplasm is present; multiple cerebral tumors are uncommon. They vary in size; occasionally they attain the size of a man's fist. Neoplasms situated upon or near the surface of the brain generally give rise to circumscribed projections, in the neighborhood of which the gyri are generally flattened and the sulci obliterated. Under such circumstances marked displacement of the falx cerebri toward the opposite cerebral hemisphere may take place in consequence of pressure. Compression and displacement of neighboring structures may result also from neoplasms situated in the depth of the brain. Not rarely internal hydrocephalus has developed in consequence of pressure and stasis. It is not always easy to recognize tumors of the brain with the unaided eye. Suppurating tubercles may, for instance, readily be mistaken by an inexperienced person for ordinary cerebral abscesses, or vascular gliomata may be confounded with a hemorrhagic focus in the brain, particularly if blood-vessels have ruptured in the tumor. Most varied forms of neoplasm occur in the brain. Gliomata and sarcomata are among the most common. In addition, there may be mentioned psammomata, myxomata, carcinomata, melanomata, and cholesteatomata. Only in a few instances have papillomata, fibromata, lipomata, enchondromata, osteomata, dermoid cysts, other cysts, and angiomata been found. *Hyperplasia of the pituitary body—pituitary struma*—may also be included among central tumors.

Symptoms and Diagnosis.—A tumor of the brain develops at times without symptoms, and is discovered quite accidentally on post-mortem examination—*latent tumor of the brain.* In other instances it gives rise to **general cerebral symptoms**, and in still others also to **focal symptoms.** The seat of the neoplasm can be determined only from the latter. Among the general cerebral symptoms are especially *headache*, *vomiting*, and *papillitis.* But rarely will a neoplasm develop in the brain without *headache.* At times this is confined to a circumscribed area of the skull, while at other times it involves the entire skull or is deep-seated. Generally, no definite conclusion as to the seat of the brain-tumor can be reached from the situation of the headache. The pain is at times described as beating, at other times as boring or crushing. It often occurs in paroxysms, and occasionally attains such a grade of severity that the patient loses his senses or is seized with unconsciousness and general convulsions. At times *percussion of the head* in certain places gives rise to severe pain, although this symptom also can scarcely be availed of in localizing the seat of the brain-tumor. Patients with tumor of the brain frequently suffer from *vomiting*. It is noteworthy that the emesis often occurs without preceding nausea, and that it takes place with exceedingly slight effort. Generally the vomiting occurs in paroxysms, and often it coincides with attacks of headache. Should it persist for days, and be repeated at short intervals, the patients often enter into a state of alarming exhaustion. ***Papillitis*** is a symptom of unusual importance in the diagnosis of brain-tumor, and ophthalmoscopic examination of the fundus of the eye should never be omitted when a suspicion of the presence of a brain-tumor exists. Visual disturbances may be wholly wanting in spite of developed papillitis, while in other instances progressive impairment of vision (amblyopia) occurs, and this may gradually progress to blindness (amaurosis).

In all probability papillitis (also known as choked disc) is the result of progressive increase in intracranial pressure, in consequence of which cerebrospinal fluid is forced between the sheaths of the optic nerve, so that the arterial supply to, as well as the venous discharge from, the retina is interfered with. According to another view, the choked disc is considered the result of an inflammatory process, whence the designation papillitis. The condition can be readily recognized with the ophthalmoscope, as the optic papilla projects like a bulb into the interior of the eye and presents a grayish or grayish-red appearance. The contour of the optic papilla is indistinct and the retinal arteries are strikingly narrow, while the retinal veins are distended and exceedingly tortuous. Papillitis may slowly be transformed into optic atrophy. Further, sudden variations occasionally occur in the development of papillitis in the course of the disorder, and at times the condition disappears entirely.

Papillitis alone is not distinctive of tumor of the brain, as it occurs also in association with meningitis, abscess of the brain, hydrocephalus, and at times even with cerebral hemorrhage. It occurs also in cases of nephritis and lead-poisoning.

Less diagnostic importance is to be attached to the remaining general cerebral symptoms. Complaint is often made of *vertigo*, and particularly in the presence of neoplasms in the cerebellum the patient staggers like a drunken man—so-called cerebellar ataxia. Occasionally the patient exhibits a tendency to fall constantly toward the same side. The pupils are frequently unequal, and react sluggishly to light, [illegible] to sensory irritation. The pulse often is strikingly slow, [illegible] consequence of irritation of the center for the vagus [illegible] Not rarely slowing of the pulse occurs [illegible] Often complaint is made of [illegible] Frequently the patients complain of vague [illegible] and upon superficial investigation may [illegible] nervous or hysterical. Not rarely [illegible] The patient may become apathetic and torpid [illegible] a marked psychopathy. Frequently attacks of [illegible] occur, which occasionally may persist for days [illegible] and talk incoherently, and pursue a sort of dream-life, from which they emerge only from time to time. While in such states of confusion they manipulate their genitalia with relative frequency, without any indication of causing irritation in the genital sphere. At times *general clonic muscular spasm* occurs, so that the condition may readily be confounded with *epilepsy*. *Polyuria, albuminuria,* and *glycosuria* are among the less common occurrences, and depend upon involvement of certain points upon the floor of the fourth ventricle.

All general cerebral symptoms are susceptible of great variation in regard to their occurrence and their intensity, probably in consequence of the varying degree of vascularity of the new-growth and the variations in the cerebral pressure. It is by no means rare for the symptoms of a cerebral tumor to be limited to general cerebral manifestations, and under such circumstances the situation of the new-growth remains involved in doubt. With regard to the *seat of a tumor of the brain,* the *focal symptoms* present alone are distinctive in accordance with the diagnostic conditions.

The *anatomic character of a tumor of the brain* can but rarely be determined with certainty during life. If tuberculosis or syphilis have been an antecedent condition, the supposition of a tuberculoma or of a gumma is most probable. In the presence of metastatic new-growths in the brain the character of the primary neoplasm often is decisive as to the nature of the cerebral tumor. Generally it is impossible to decide whether a new-growth is situated within the brain itself or has originated from the meninges or the bones of the skull. Peripheral pressure-paralysis of cerebral nerves at the base of the brain is especially indicative of a meningeal neoplasm.

The *duration of a tumor of the brain* may extend over several years. The majority of cases naturally terminate fatally within the first year of the disease. Occasionally death takes place suddenly amid symptoms of an *apoplectic attack,* and under such conditions recent extravasations of blood into the tumor-mass are found at times, though not frequently. In other cases death results from *increasing cerebral pressure* in the presence of unconsciousness or general clonic muscular spasm, or from paralysis of the vagus. Less commonly a cerebral tumor is complicated by *abscess of the brain* or *meningitis,* which causes death.

Prognosis.—The prognosis of tumor of the brain is serious. Attempts have recently been made to remove neoplasms in the brain by surgical means after opening the skull, but the prognosis has scarcely been improved thereby.

Treatment.—**Causative treatment** is applicable only in the presence of gummata. Under such conditions inunctions of mercurial ointment and the internal administration of potassium iodid will be indicated.

Neoplasms whose seat can be accurately localized from the focal symptoms have in a number of instances been successfully removed by **operation.** The tumors under such circumstances have generally been situated in one of the central convolutions, and have given rise to monoplegia or hemiplegia and clonic muscular spasm in typical distribution. A successful result is by no means certain, even when the localization has proved correct, for at times the tumor is too extensive to permit of removal, or fatal hemorrhage, meningo-encephalitis, and septicemia or sudden collapse occurs. After successful operation paralysis or epileptic attacks at times remain. In cases of inoperable tumor of the brain **palliative trephining** of the skull also is reported to have yielded material relief. Symptomatically treatment must frequently be directed against the headache and the vomiting. For this purpose **phenacetin** (1.0—15 grains—thrice daily), **antipyrin** 1.0—15 grains—thrice daily), **sodium salicylate** (1.0—15 grains—as often as four times daily), and in marked cases **subcutaneous injections of morphin** may be recommended. The application of an **ice-bag** to the head usually mitigates the severity of the headache. Some patients obtain material relief from their symptoms from the employment of **bromids.** A **proper mode of life** is of importance, and alcoholics and dietetic excesses are particularly to be avoided. Daily evacuation of the bowels also should be secured. Sexual intercourse should be interdicted.

CEREBRAL PARASITES.

Cerebral parasites are of rare occurrence. Those that have been observed are especially the **Cysticercus cellulosæ** (*the measle of pork*) and **echinococci** (*the measle of the tapeworm of the dog*). The *Cysticercus cellulosæ* may develop either from the meninges, and most frequently from the tissue of the pia, or from the brain-tissue itself, particularly its gray matter. At times it occurs in the subarachnoid space or in one of the ventricles of the brain as a free cyst. The Cysticercus racemosus, which is a multilocular cyst, is rarely observed. At times the cerebral cortex is covered with hundreds of vesicles, which have in part become embedded in the surface of the brain. The individual cyst varies in size, which at times attains that of an apple. After from three to five years the cysticerci generally die and undergo caseation and calcification, so that they might readily be confounded with tuberculous nodules if they did not contain the hooklets readily recognizable and demonstrable on microscopic examination.

The manner in which cysticerci gain entrance into the interior of the skull is unknown. At times they are *unattended with symptoms,* while at other times they give rise to symptoms of a *cerebral* *tumor*, or to a *psychopathy*, or to *epilepsy*. That the cerebral symptoms present are due to cysticerci may be suspected if the patient harbors a tapeworm (Tænia solium), or if cysticerci can be discovered in the subcutaneous connective tissue or in the retina. As *treatment* is futile, the *prognosis* is unfavorable. Occasionally death occurs suddenly.

Echinococci of the brain generally occur in the form of a unilocular cyst. Less commonly the echinococcus-cysts are multiple. The cysts may be sterile or they may contain daughter-cysts. A cyst may at times attain the size of the entire cerebral hemisphere. Generally echinococcus-cysts of considerable size give rise to the symptoms of a *cerebral tumor*, with a preponderance of *epileptic convulsions*. At times they rupture externally, discharging a number of daughter-cysts, and undergoing gradual recovery. They cannot be removed by means of internal remedies. In a number of instances they have been *removed by surgical means*.

ANEURYSMS OF THE CEREBRAL ARTERIES.

Aneurysms of the cerebral arteries develop most frequently upon the vessels of the circle of Willis, and occur with especial frequency upon the middle cerebral artery or the artery of the fossa of Sylvius, and next in frequency upon the basilar artery. At times aneurysms have developed upon several cerebral arteries. They sometimes attain the size of a hen's egg. All of the coats of the artery are involved in the saccular dilatation of the vessel, and the condition is, accordingly, one of true aneurysm. The disorder occurs *more commonly in men* than in women, and not rarely in young persons. *Injuries of the skull, excessive indulgence in alcohol*, and *syphilis* are said to be causes, although frequently no predisposing factor can be demonstrated. At times the condition is *unattended with symptoms*. At other times *general cerebral symptoms*, and with especial frequency throbbing headache, *mental disturbance* or *epilepsy*, or, in consequence of pressure upon adjacent nerves and portions of the brain, *basal peripheral paralysis of cerebral nerves* or *cerebral focal symptoms* are present; but these are not peculiar to this disorder. Death often occurs suddenly amid the symptoms of an apoplectic attack if the aneurysm has ruptured and a copious extravasation of blood has taken place in the subarachnoid space, and often also destruction of brain-tissue.

Only in rare cases is the **diagnosis** possible during life. Occasionally cardiac-systolic vascular murmurs have been heard between the mastoid process and the muscles of the neck.

The **prognosis** is unfavorable.

The question of **treatment** will arise but rarely, on account of the difficulty in diagnosis, and the indications are generally for the relief of troublesome symptoms.

HYDROCEPHALUS.

Hydrocephalus occurs by reason of its origin in two forms: *congenital* and *acquired.*

CONGENITAL HYDROCEPHALUS.

Anatomic Alterations.—Congenital hydrocephalus is generally *ventricular* or *internal,* less commonly *intermeningeal* or *external.* In the first event serous fluid has collected in the ventricles of the brain in an amount reaching on the average a liter, but at times having been as much as 14 liters. The cerebral ventricles under such circumstances naturally represent large chambers, and the foramen of Monro is often converted into a large orifice. The basal ganglia are flattened in consequence of compression, and often the entire cerebral mass is so attenuated that it resembles a vibratile sac filled with fluid. The extensive increase in size of the brain is attended also with a corresponding increase in the size of the skull.

Hydrocephalus often is associated with other *malformations,* as, for instance, meningocele, encephalocele, syringomyelia, harelip, cleft palate, or club-foot.

Etiology.—The causative factors of congenital hydrocephalus are often involved in doubt. Among those that have been mentioned are alcoholism and syphilis in the parents, injuries during pregnancy, maternal impressions, and diseases of the uterus, the last of which is believed to cause circulatory disturbances within the skull of the embryo during pregnancy. Occasionally several children in the same family have presented congenital hydrocephalus.

Symptoms and Diagnosis.—At times the head of the embryo becomes so greatly increased in size in consequence of hydrocephalus, even before parturition, that delivery is not possible without *dismemberment of the fœtus,* or the head is subjected to such compression during delivery that the *skull is torn,* and hydrocephalic fluid escapes. Not rarely the children are born prematurely, and are stillborn. Some children present scarcely any peculiarity immediately after birth, but they soon attract attention by reason of the progressive increase in the **size of the head,** and within a short time all doubt is removed that the condition is a morbid one. The circumference of the head, which measures from 35 to 40 cm. in the newborn, and at the end of the first year of life reaches 45 cm., and at the period of puberty 50 cm., may be quadrupled. At the same time the **sutures of the skull** appear as wide fissures, and the **fontanels**

acquire enormous size. When ossification subsequently takes place this is generally effected through the formation of supernumerary bones. The **bones of the skull** are exceedingly thin, and here and there compressible. The skull is at times so translucent that the flame of a candle can be seen through it. The face, in comparison with the enormous size of the skull, presents the appearance of a small appendage. At times **vascular murmurs** are audible upon the skull. Dilated and tortuous cutaneous veins often are present in the temporal regions. **Nystagmus** is not rarely present. **Papillitis, atrophy of the optic nerves,** and **amaurosis** also may develop. **Epileptic attacks** occur not rarely. **Muscular contractures, muscular spasm,** and **exaggeration of the tendon-reflexes** likewise are often present. In the vertical posture the child can scarcely balance the enormous head upon the body, and it usually assumes the recumbent posture. It learns to walk only with difficulty and late, and it readily falls forward. The gait for a long time exhibits great uncertainty and marked swaying. The **mental faculties** often suffer, and idiocy may develop. Many children die in consequence of excessive cerebral pressure. Others develop slowly, and reach advanced age.

Prognosis.—The prognosis of congenital hydrocephalus is unfavorable, for success cannot be secured with internal remedies, and the application of operative measures is not sure to bring relief, and is likewise not unattended with danger.

Treatment.—**Potassium iodid, ointments of iodin** or **applications of tincture of iodin to the skull** generally are equally useless with the employment of **diuretics, drastics,** or **diaphoretics. Compression of the skull** with strips of adhesive plaster or bandages is likewise unattended with noteworthy improvement. In a number of cases **puncture of the ventricles,** and even **drainage,** has been attempted, and in isolated instances with success. **Lumbar puncture** of the spinal subarachnoid space will scarcely yield permanent cure.

ACQUIRED HYDROCEPHALUS.

Etiology.—Acquired hydrocephalus is frequently of **inflammatory origin.** It is observed to occur with especial frequency in connection with purulent and tuberculous meningitis. **Stasis,** both general, in connection with diseases of the heart and the respiratory organs, and local, not rarely gives rise to acquired hydrocephalus. Among the local causes of stasis are neoplasms that compress the great veins of Galen, and adhesions and occlusion of the lymphatics and the blood-vessels in the sequence of meningitis. At times the condition is one of *cachectic hydrocephalus,* such as may develop in connection with carcinoma, diseases of the kidneys, and pulmonary tuberculosis. In some cases of hydrocephalus the causative factors remain undetermined. **Syphilis, alcoholic abuse,** the influence of **great heat and excessive mental activity** are said to give rise to hydrocephalus. Hydrocephalus is not rarely observed in *rachitic children,* and children in general are especially predisposed to hydrocephalus.

Anatomic Alterations.—In cases of *external* or *intermeningeal hydrocephalus* the subarachnoid spaces contain an excessive amount of fluid, and at times the cerebral cortex appears flattened and attenuated. *Internal* or *ventricular hydrocephalus* is characterized by the presence of an excessive amount of fluid in the ventricles of the brain. The fluid is clear, or turbid and flocculent, and at times hemorrhagic if the condition is of inflammatory origin. Large accumulations of fluid give rise to compression and flattening of the basal ganglia, distention of the ventricles of the brain, compression and attenuation of the entire brain-mass, as has already been detailed in the description of congenital hydrocephalus. At times the fluid ruptures through the brain, relatively with greatest frequency through the convexity.

Occasionally fluid has accumulated only in certain portions of the cavities of the brain, as, for instance, only in one horn, or only in a single ventricle, and has caused abnormal dilatation of the affected parts. Such a condition is designated *internal saccular* or *circumscribed hydrocephalus.*

Symptoms and Diagnosis.—Clinically a distinction must be made between **acute** and **chronic hydrocephalus.** *Acute hydrocephalus* occurs especially in consequence of purulent or tuberculous meningitis, and probably contributes in part to the symptoms of abnormally increased cerebral pressure. Distinctive symptoms do not occur, and accordingly the recognition of the condition is rather a matter of inference than of accurate diagnosis. *Chronic hydrocephalus,* when it occurs in children under the age of seven years, gives rise to **enlargement of the head** like that described in association with congenital hydrocephalus, and all of the other symptoms also agree with those of congenital hydrocephalus. In children after the seventh year of life, and particularly in adults, the ossified cranium offers sufficient resistance to the pressure exerted by the hydrocephalic fluid, and, accordingly, deformity of the skull does not take place. Hydrocephalus may, however, occur even in the presence of a skull of small size, as, for instance, in cretins. The patient often complains of headache and vertigo. Occasionally attacks of vomiting, slowing of the pulse, and loss of consciousness, with other signs of increased cerebral pressure, occur. Mental development often suffers, and dementia results. Epileptic convulsions, paralysis, muscular twitching, spasm, and contracture also are not uncommon. Papillitis and optic atrophy occur, so that the clinical picture may be confounded with that of a cerebral tumor. Occasionally death takes place suddenly, amid the symptoms of an apoplectic attack, or with epileptic convulsions; or at other times, long-continued comatose conditions develop, which terminate fatally, although the patient may attain advanced life. Occasionally spontaneous recovery has been observed in consequence of rupture and discharge of the hydrocephalic fluid externally, the fluid at times escaping for years through the nose, the orbit, the ear, or the mouth.

Prognosis and **treatment** are the same as in cases of congenital hydrocephalus.

HYPERTROPHY OF THE BRAIN.

Extremely little of a definite nature is known with regard to hypertrophy of the brain. The condition is assumed to exist if on opening the skull the brain bulges from the cranial cavity and can be replaced only with difficulty, while at the same time cerebrospinal fluid is absent, the surface of the brain is flattened and bloodless, and the ventricles of the brain appear contracted. It is unreliable to consider the weight of the brain in the anatomic diagnosis of cerebral hypertrophy, because of the wide variations even under normal conditions. Generally the demarcation between white and gray brain-matter is indistinct, and often the consistence of the brain-tissue is increased, so that it is suggestive of coagulated albumin or rubber. Most commonly both cerebral hemispheres are involved in hypertrophy. Less commonly the hypertrophy is circumscribed, being limited, for instance, to the cerebellum or the pons or to certain other portions of the brain. In children, however, enlargement of the skull in its posterior portion, or in adults roughness upon the inner surface and attenuation of the cranial bones, are important. A distinction must be made between *congenital* and *acquired hypertrophy of the brain*. Congenital hypertrophy of the brain is occasionally *inherited*, and often occurs in *rachitic children*. Acquired hypertrophy of the brain has been attributed to *alcoholic abuse*, *lead-poisoning*, *syphilis*, *excessive mental activity*, and *injuries of the skull*.

Hypertrophy of the brain is at times *unattended with symptoms*. If it occurs in children, *increase in the size of the skull* may take place, and this may readily be confounded with a hydrocephalic skull, although the enlargement involves rather the posterior portion of the skull than the frontal region. In older children and in adults the change in the size of the skull does not occur. The disease can, therefore, never be recognized with certainty in the latter, as under the best of circumstances it gives rise to general cerebral manifestations. Among these, headache, vertigo, vomiting, epileptic attacks, and muscular contractures and spasms may be mentioned. The mental faculties are occasionally developed to an almost abnormal degree at first, but subsequently a condition of progressive dementia often appears. The disorder may persist for many years, and occasionally terminates suddenly in the presence of coma, epileptic convulsions, or an apoplectic seizure.

The **prognosis** is unfavorable, as the **treatment** can be directed only to the relief of prominent symptoms.

ATROPHY OF THE BRAIN.

Atrophy of the brain is of but slight clinical interest, because it can only rarely be recognized. Occasionally the condition is one of *congenital atrophy of the brain*, involving either the entire organ or only one hemisphere or certain portions of the brain. *Infantile hemiatrophy of the brain* is not rarely attended with paralysis, atrophy, and contracture of the extremities upon the opposite side of the body. The growth of the bony parts of the affected extremities is retarded. Often epileptic convulsions occur, and the children are not rarely idiotic. Life may naturally be preserved for many years. Among the varieties of *acquired atrophy of the brain*, *senile atrophy of the brain* should be mentioned first, and to which loss of mental power, tremor, and readiness of fatigue in the aged have been attributed. Closely related to this variety is *marantic atrophy of the brain*, which has been observed in the sequence of long-continued diseases, particularly carcinoma. *Compression-atrophy of the brain* develops in the sequence of meningitis, hemorrhage, neoplasms, and the like. *Toxic atrophy of the brain* has been observed in drunkards, workers in lead, and smokers of opium. The surface of a brain the seat of general atrophy is separated from the inner surface of the skull by an interval of unusual width, and the ventricles of the organ are dilated; while an unusually large amount of cerebrospinal fluid is present in the subarachnoid spaces and in the ventricles of the brain.

DIFFUSE SCLEROSIS OF THE BRAIN.

[illegible] proliferation of neuroglia, [illegible] a firm, rubber-like [illegible]

Etiology. [illegible] and excessive [illegible]

[illegible] symptoms [illegible] disturbances [illegible] predomi- [illegible] paralysis, [illegible] paralysis of the [illegible] occur. Not [illegible] anesthesia, and [illegible]

[illegible] and at

times terminates suddenly amid the symptoms of an apoplectic attack.

The **diagnosis** can scarcely ever be made with certainty.

The **treatment** is purely symptomatic.

DISEASES OF THE CEREBRAL MENINGES.

THROMBOSIS AND INFLAMMATION OF THE SINUSES OF THE BRAIN.

Etiology.—Occlusion of the sinuses of the brain by fibrinous deposits or thrombi results from the same causes as are operative in other veins, and accordingly a distinction can be made between *inflammatory, marantic, traumatic,* and *compression-thrombosis of the cerebral sinuses.*

Inflammatory thrombosis of a cerebral sinus is attended at first with inflammatory alterations in the wall of the sinus, and it is these that render possible the deposition of fibrinous precipitates from the blood. Often the inflammation of the wall of the sinus has resulted by **extension from adjacent disease,** and **diseases of the petrous bone and the internal ear** give rise to the condition with especial frequency. *Meningitis* and *cerebral abscess* also are occasionally complicated by inflammation and thrombosis of the sinuses of the brain. At times the inflammatory foci are situated more remotely, and the exciting agents of inflammation are conveyed to the sinuses of the brain through the intermediation of the blood-current and the lymph-paths. Such a condition has been observed in association with *facial erysipelas, injuries of the face or the skull,* the development of *furuncles and of abscesses on the face and upon the gums,* in the presence of *eczema of the face* and of *inflammation of the orbit and the nasal cavities.* Sinus-phlebitis and sinus-thrombosis develop at times also in the course of *infectious diseases,* and most commonly in connection with septicopyemia. The presence frequently of bacteria (Streptococcus and Staphylococcus pyogenes, Bacterium coli) in the fibrinous deposits and their conversion into puriform septic masses are distinctive of inflammatory thrombosis.

Marantic thrombosis of a cerebral sinus may develop in the sequence of debilitating disease of any kind; in children, for instance, after chronic diarrhea, and in adults in the course of carcinoma, pulmonary tuberculosis, protracted suppuration, chronic diarrhea, and the like.

Traumatic thrombosis of a cerebral sinus occurs but rarely. It has been observed in isolated instances following injuries of the skull and the cerebral sinuses.

Compression-thrombosis occurs occasionally in association with neoplasms of the brain, meningitic thickening, and allied conditions.

Thrombosis of a cerebral sinus by continuity further is worthy of mention. The condition may result from the extension of a thrombosis of the superior vena cava or of the internal jugular vein upward to the transverse sinus or the inferior petrosal sinus.

Anatomic Alterations.—In the presence of sinus-thrombosis the diseased sinus on incision will be found totally occluded or merely constricted by fibrinous masses, so that a distinction is commonly made between totally *obstructive* and *mural sinus-thrombosis.* In contradistinction from the coagula that are without significance, the thrombotic masses adhere firmly to the wall of the sinus and are characterized by their denser consistency and brownish-red or grayish-red color. On transverse section their laminated structure can frequently be recognized. The conditions are different in the presence of *inflammation of the sinuses of the brain* and *infectious sinus-thrombosis,* for under such circumstances the thrombotic formation consists of a smeary, grayish or greenish, purulent mass, which at times gives off an offensive odor, and the wall of the sinus appears soft and friable. Often **metastatic abscesses** are present in various organs, particularly the lungs, obviously from the conveyance of infectious portions of the thrombus into the distribution of the internal jugular vein, and further toward the heart. The **superior longitudinal sinus** and the **transverse sinus** are most frequently the seat of thrombosis. As stagnation occurs in the venous blood-stream when a sinus of the brain is occluded, signs of hypostatic hyperemia in the related veins may be expected. The symptoms of stasis will be most marked when the thrombus obstructs both transverse sinuses and inferior petrosal sinuses, and has further extended into the internal jugular veins. Under such circumstances there may be cerebral hemorrhage, hydrocephalus, and hemorrhagic discoloration of the cerebrospinal fluid.

Symptoms, Diagnosis, and Prognosis.—Thrombosis of the cerebral sinuses is at times unattended with noteworthy symptoms, and therefore remains unrecognized—*latent thrombosis of the cerebral sinuses.* At other times, at least, **general cerebral manifestations** indicate that morbid processes have taken place within the skull. Among these are coma, general clonic convulsions, inequality of the pupils, muscular contractures, and paralysis. Occasionally sinus-thrombosis is attended with the symptoms of purulent **meningitis**, and not rarely meningitis, together with thrombophlebitis of the cerebral sinuses, is encountered in association with inflammatory thrombosis. Frequently the disorder just named is concealed behind the symptoms of **septicopyemia,** and it gives rise with especial frequency to **pulmonary abscess**, for which no other cause can be elicited. The *symptoms of pulmonary* *infarction* also are sometimes dependent upon sinus-thrombosis, if portions of a non-infective sinus-thrombus have become detached and have gained entrance into the right heart and thence into the distribution of the pulmonary artery through the intermediation of the internal jugular vein and the superior vena cava.

Thrombosis of the cerebral sinuses is susceptible of recognition only when **signs of stasis or of deranged circulation in the veins of the face or the neck** have made their appearance. These vary in accordance with the cerebral sinus affected. In the presence of *thrombosis of the superior longitudinal sinus* all of the cutaneous veins between the anterior fontanel, the temporal and the auricular regions are extremely tortuous and dilated. Cyanosis of the skin develops in the distribution of the anterior facial vein. Not rarely epistaxis occurs. Profuse sweating of the forehead, the head, the neck, and the chest has also been observed. In infants the anterior fontanel is depressed and becomes tense again only when hydrocephalus has developed in connection with the sinus-thrombosis.

Thrombosis of the transverse sinus is occasionally attended with **diminished fulness of the external jugular vein,** which delivers its blood more readily to the internal jugular vein if this no longer receives blood from the occluded transverse sinus. At times **edema behind the auricle and over the mastoid process** develops if the thrombosis has extended from the transverse sinus through the emissary vein of the mastoid process to the posterior auricular veins.

In the presence of *bilateral thrombosis of the transverse sinus* the same symptoms may develop as attend thrombosis of the superior longitudinal sinus in consequence of retrograde stasis.

Thrombosis of the cavernous sinus gives rise to symptoms of unilateral or bilateral **stasis about the eye,** accordingly as the thrombosis is unilateral or bilateral. **Acute protrusion of the eyeball (exophthalmos)** develops in consequence of hypostatic hyperemia of the retrobulbar veins. **Edema of the eyelids and the conjunctiva** develops. Ophthalmoscopic examination discloses **marked tortuosity and over-distention of the retinal veins, papillitis, retinal edema,** at times **thrombi in some of the retinal veins,** and **impairment or even loss of the power of vision.** These disturbances are explained by the fact that the ophthalmic vein empties into the cavernous sinus. As, however, the ophthalmic vein also communicates with the anterior facial vein, **edema of the face** occasionally develops. To the symptoms mentioned frequently **paralysis of the ocular muscles, neuralgia in the distribution of the first division of the fifth nerve,** at times also **trophic disturbances about the eye** become superadded, because the nerves in question pass through the cavernous sinus and are readily compressed and paralyzed by the edema of the tissues of the sinus.

The symptoms of thrombosis of the cavernous sinus occur also in association with *thrombosis of both transverse and inferior petrosal sinuses*, because these receive the blood of the cavernous sinus.

Distinctive symptoms of obstructed venous circulation resulting from thrombosis of the remaining cerebral sinuses are not known. Thrombosis of a cerebral sinus is always a serious disorder, which occasionally terminates fatally within a few days. At times it may persist for several weeks with numerous variations in the symptoms. It is noteworthy that the condition is not rarely attended with fever, even if the thrombosis is not infective. It has not been demonstrated with certainty whether it is possible for a thrombus to be gradually absorbed or to become again patulous after penetration by new blood-vessels, and recovery thus result. Death occurs not rarely in the presence of progressive unconsciousness or of general clonic muscular spasms, or amid the symptoms of meningitis, cerebral abscess, or septicopyemia.

Treatment.—No remedy is known that is capable of causing absorption of a thrombus, so that only **symptomatic treatment** is applicable. In the presence of *thrombophlebitis* of a cerebral sinus **operative treatment** has been undertaken recently with marked success, the diseased sinus being opened, the suppurating thrombus evacuated, and hemorrhage controlled by plugging the wound with iodoform-gauze. In order to prevent dissemination of infectious material the internal jugular vein has first been ligated in two places, and the vessel divided between these. Operation, however, will, as a rule, yield complete success only if signs of general septicopyemia and metastatic purulent foci have not yet developed, whence it is clear that the operation should be resorted to at the earliest possible moment.

INTERNAL HEMORRHAGIC CEREBRAL PACHYMENINGITIS.

Anatomic Alterations and Etiology.—Quite often, on post-mortem examination, thin, veil-like membranes are encountered upon the inner surface of the cerebral dura mater which are traversed by recent or old hemorrhages, and in the latter event have often acquired a yellowish or icteric appearance in consequence of transformation of the hemoglobin of the emigrated red blood-corpuscles into hematoidin. The size, the number, and the consistency of the membranes are susceptible of great variation. Often free hemorrhage is found in addition, and the impression is gained that these have occurred first, and subsequently given rise to inflammation and the formation of membrane. According to another view, the formation of membrane is considered the earliest process, and the hemorrhage is thought to result from rupture of the numerous small blood-vessels in the membrane.

The alterations described are encountered in association with all possible diseases, with especial frequency in connection with infectious diseases, conditions of chronic stasis, and profound anemia, and they are only of anatomic interest, as by reason of their delicacy they cause no cerebral disturbances. At times, however, the alterations described give rise to the development of extensive blood-cysts, which have been designated *hematomata of the cerebral dura mater.* These develop with especial frequency upon the convexity of the brain, enclosing one, sometimes both hemispheres with a thick layer of blood, and by pressure they give rise to marked flattening, and even to a depression upon the surface of the brain. Occasionally free blood has ruptured into the subarachnoid space, and has even given rise to laceration of brain-tissue. On incision of such a hematoma it will be found to consist of superimposed layers of membrane, between which more or less old and correspondingly altered blood is present. The oldest layers of blood lie nearest the inner surface of the dura mater, while the most recent are in contact with the surface of the brain. Generally the entire blood-cyst can be detached from the inner surface of the dura without serious difficulty. *Microscopic examination* discloses the presence of degenerated red blood-corpuscles in all possible stages of transformation to a yellowish granular, flocculent, or crystalline blood-pigment, between the individual layers. Also the membranes themselves, the oldest of which occasionally have acquired a dense, fibrous character, are abundantly infiltrated with precipitated hemoglobin.

The development of hematomata in all probability takes place in such a manner that hemorrhage occurs from the numerous delicate blood-vessels of the most recently formed membrane, and this blood in turn is enclosed by new membrane, *Drunkards* present hematomata with especial frequency. These occur also with frequency in the *insane.* Occasionally they develop in the sequence of **traumatism.** **Syphilis** also is believed to cause the formation of hematomata.

Symptoms and Diagnosis.—Hematomata of the dura mater give rise to symptoms of increased cerebral pressure and **general cerebral phenomena** in consequence of crowding and increase in the pressure within the skull. Headache and vertigo are especially constant symptoms; in addition, inequality and contraction of the pupils, slow pulse, at times papillitis, with especial frequency, however, coma, occur. At times the patients remain for days or even for weeks in a dreamy, somnolent state, muttering incoherent words. Conjugate deviation of the head and eyes has often been observed. The bodily temperature is not rarely elevated. **Local cerebral symptoms** also may occur, particularly

arm in consequence of irrita-
member, contractures, con-
or total paralysis when the
occasionally the patient recovers
extravasated blood is absorbed and
It is, however, distinctive that
described occur as soon as
the hemorrhage is considerable,
in the presence of symptoms
symptom is known that is peculiar
diagnosis can scarcely ever be
agnosis the etiology especially

dural hematoma is grave, as
progress and to be attended eventu-

were reasonably certain, **open-
ation (trephining)** and evacuation
indicated. In the absence of
ment must suffice, and this will
an *ice-bag* to the head and an
0 : 200—75 grains : 6½ fluid-
thrice daily) is largely used.

CEREBRAL MENINGES.

the *epidural space*; that is,
wall and the outer aspect of the
blood in this situation is possi-
has separated the dura from the
condition generally results from
such hemorrhage occurs in the
contracted pelvis or compression
bones of the fetal head are sub-
blood-vessels are thereby lacer-
marked compression of the brain,
dead at birth. Occasionally they
or they recover completely,
hemiplegia (p. 67). Epidural
internal hematoma of the cerebral
on the inner aspect of the dura
dural (arachnoid) hemorrhage.
capillary space. The condi-
of internal pachymeningitis as
p. 83). *Subarachnoid hemorrhage*
and the pia, in that mesh-worked
spinal fluid is contai Such hemor-

rhage may result, among other causes, from ruptured aneurysms of the cerebral arteries and injuries of the middle meningeal artery. Occasionally extravasations of blood rupture from the subdural space or from the brain into the subarachnoid space. The hemorrhage may be considerable; it occasionally escapes in part into the spinal canal, and causes marked compression and flattening and at times even laceration of the brain. The symptoms during life are those of an acute increase in cerebral pressure generally, with the symptoms of an apoplectic attack (coma, slowing of the pulse, irregular respiration, contraction, and inequality of the pupils). In a number of instances recovery from injuries of the meningeal artery has been brought about by *opening the skull* and removing the extravasated blood. Otherwise an *ice-bag* and *stimulants* should be prescribed. *Subpial extravasations of blood* are situated between the pia and the surface of the brain, and can take place only if the pia is forcibly detached from the cerebral cortex. They generally result from preceding cerebral hemorrhage.

V. DISEASES OF THE SYMPATHETIC NERVE.

LITTLE of a definite nature is known with regard to the diseases of the sympathetic. It is true a number of morbid manifestations have been attributed to diseases of the sympathetic, in explanation of which no alterations can be demonstrated in any other portion of the nervous system, but these are more or less arbitrary assumptions. We shall confine ourselves here to the paralytic and irritative states in the distribution of the cervical sympathetic.

PARALYSIS OF THE CERVICAL SYMPATHETIC.

Symptoms.—Paralysis of the cervical sympathetic in human beings is attended with the same *symptoms* as are observed in animals after division of the cervical sympathetic. In consequence of paralysis of the vasomotor nerves dilatation of the blood-vessels, together with marked **redness**, of the face, the neck, and the upper portion of the chest down to the nipple upon the affected side takes place. These alterations naturally may recede after a time, and be replaced by diminished fulness of the vessels. At the same time profuse **sweating** occurs. Paralysis of the unstriated muscle of Müller in the orbit gives rise to **retraction of the eyeball and narrowing of the palpebral fissure.** The pupil is **contracted,** and reacts but sluggishly to light and not at all to sensory irritation. Although atropin causes dilatation of the pupil, this will be slighter than upon the healthy side. **Trophic disorders** may gradually arise, as, for instance, atrophy of one side of the face.

Etiology.—The *causative factors* for paralysis of the sympathetic are generally of **traumatic nature,** for instance, punctured, gunshot and operative wounds of the neck, compression by enlarged lymphatic glands, inflammatory processes involving the connective tissue of the neck, enlarged thyroid gland, fracture and luxation of the cervical vertebræ, disease of the cervical cord or of the lower portion of the brachial plexus (Vol. I., p. 499), and pulmonary tuberculosis if adhesions exist between the **apex** of the diseased lung and the cervical sympathetic.

The **prognosis** depends upon the removability of the causative factors.

Treatment.—**Galvanization** of the cervical sympathetic has been recommended in treatment. In addition, **operative intervention** must be taken into consideration.

IRRITATION OF THE CERVICAL SYMPATHETIC.

Symptoms.—Irritation of the cervical sympathetic is primarily attended with **dilatation of the pupils—spastic mydriasis.** Often the power of accommodation also is impaired. Occasionally **protrusion of the eyeballs** and **widening of the palpebral fissure** occur in consequence of irritation of the muscle of Müller. As a result of vasomotor spasm the **carotid** and **temporal arteries at times** are contracted, and the side of the face and the neck are pale and cool; and sweating may be absent. **Trophic disorders** may develop within a short time, and give rise to disfiguring unilateral atrophy of the face.

Etiology.—The same *causative factors* that are responsible for paralysis of the cervical sympathetic may give rise also to irritation, and occasionally irritative and paralytic states alternate with each other.

With regard to **prognosis** and **treatment** the same statements are applicable as were made concerning paralysis of the sympathetic.

VI. CENTRAL NEUROSES.

THOSE nervous disorders are designated neuroses for which hitherto it has not been possible to demonstrate anatomic alterations. They are, therefore, designated also *functional nervous disorders*. *Central* neuroses include those nervous disorders whose seat is suspected to be in the central nervous system. It is the object of medical investigation to limit progressively the number of central neuroses.

CENTRAL NEUROSES IN WHICH MOTOR DISTURBANCES PREDOMINATE.

EPILEPSY.

Etiology.—In typical cases epilepsy is attended with attacks of unconsciousness and general clonic muscular spasm, which in all probability are dependent upon transitory hyperemia in the motor and purely psychic areas of the cerebral cortex. The condition is therefore designated also *idiopathic* or *true epilepsy.* The designation *symptomatic epilepsy* has been applied to cases in which neoplasms, hemorrhage, abscesses, parasites, detached splinters of bone, and the like, cause irritation of the motor centers in the central convolutions of the cerebral cortex, and as a result induce general, but frequently also localized clonic muscular spasm. Under the latter condition the disorder is designated also *cortical* or *Jacksonian epilepsy.* The characteristic feature of the latter is the fact that in many cases consciousness is preserved during the convulsive attacks, and that if general convulsions occur these always appear first in the same extremity. The remarks that follow apply only to idiopathic epilepsy.

True epilepsy is an extremely common disease that occurs rather more frequently in women than in men. Not rarely it is transmitted by *heredity,* as manifested by the occurrence of epilepsy in successive generations or by its alternation with hysteria, neurasthenia, psychopathies, or other central neuroses. Occasionally the predisposition to epilepsy is congenital. **Alcoholism in the parents,** particularly conception during a debauch, not rarely gives rise to epilepsy in the children. Occasionally **difficulty in labor** is responsible for the development of epilepsy, as, for instance, protracted labor and delivery with forceps. At times epilepsy occurs in the sequence of **infectious diseases.** Syphilis particularly is properly considered a frequent cause of the disease. **Toxic epilepsy** is worthy of especial consideration. Alcoholic excess is a frequent cause for this condition. Chronic lead-poisoning also may cause epilepsy. Some cases of epilepsy are dependent upon **injuries to the skull** and **general concussion of the body.** **Psychic influences** may also be operative. Thus, epilepsy may occur in the sequence of fright, fear, or joy, and after excessive mental activity. Some persons are attacked by epilepsy after having witnessed epileptic attacks in others, and having been deeply moved by the occurrence. Epilepsy has been observed also to develop in persons who, for one reason or another, have attempted to imitate an epileptic seizure. *Reflex epilepsy* is of considerable practical importance. This occurs in connection with diseases of organs at times far removed from the brain, an irritative influence being exerted from this point upon the brain. It is especially well known that nerves imprisoned in cicatrices may give rise to reflex epilepsy, and that the attacks can be induced by pressure upon such cicatrices, and also that cure can occasionally be effected by their removal. Polypi in the nose, the pharynx, and the ear, or foreign bodies in the nose or the ear, coprostasis, intestinal worms, phimosis, preputial calculi, uterine displacements, pregnancy, disease of the heart, and the like, may also give rise to reflex epilepsy.

Symptoms.—As a rule, epilepsy sets in *between the seventh and the twentieth year of life.* Should the first symptoms appear in adults it will be found often that special causes are operative, particularly traumatism, alcoholism, syphilis, or reflex influences. The designation *late epilepsy* has been applied to cases in which the disease begins after the fortieth year of life. Epilepsy may appear in three varieties, namely, *grave or major epilepsy, mild or minor epilepsy,* and the *epileptic equivalent.*

Grave or major epilepsy is characterized by the occurrence of *typical epileptic attacks.* These set in without demonstrable exciting cause, or they are induced by excessive psychic or physical activity, alcoholic and venereal excesses, or the like. Not rarely the occurrence of an epileptic attack is preceded by *prodromes,* among which remote and immediate must be differentiated. The *remote prodromes* frequently appear several days before the occurrence of the epileptic attack, and consist in change in character, peevishness, irritability, a tendency to anger, disturbed sleep, frequent dreams, apathy, and allied disturbances. The *immediate prodromes* are known also as the *epileptic aura,* and generally precede the epileptic attack by only a few seconds. The aura is described as sensory, motor, vasomotor, or sensorial, in accordance with the nerve-path involved in its development. The *sensory aura* may give rise to complaint of various paræsthesiæ; at times, though on the whole rarely, of a sensation as if a breath of air had been blown upon the surface, whence the designation aura (breath of air). A *motor aura* may consist in contracture, twitching, or paralysis, which not rarely begins always in the same extremity, and which in accordance with the relations of the motor cortical centers extends in regular manner and in the same order to the remaining extremities. The *vasomotor aura* is attended with states of spasm in the musculature of the vessels, and gives rise to pallor and coldness of the skin, while the *sensorial aura* is attended at times with roaring or ringing in the ears, the hearing of voices, at other times with disorders of smell or taste, generally of a disagreeable character, and at still other times with visual disturbances. In the last-named event the patient may see colors, and red with especial frequency; or he is frightened by hideous figures, which overwhelm him. Occasionally the patient is thereby impelled to acts of violence, and even to murder, and many a crime owes its commission to such processes, of which the patient has no knowledge on emerging from the epileptic attack. Such conditions are designated *pre-epileptic* (*preparoxysmal*) *insanity*.

The actual *epileptic attack* generally begins with a loud, piercing cry. The patient suddenly loses consciousness, and falls helplessly to the ground. Injuries are often incurred thereby. The unconsciousness is so profound that the patient may fall into the fire, and one or more members be burned or charred, without any perception on his part of the occurrence during the attack. At first the face and the surface of the entire body become pale, and all of the muscles of the body are thrown into tonic spasm. Within a few seconds, however, this is replaced by clonic muscular spasm, which, by reason of its wild and violent nature, gives the epileptic attack its horrible aspect. The eyeballs are rolled to and fro, and eventually are rotated inward and upward. The muscles of the face are greatly distorted. The jaws are held tightly together, and are displaced laterally, and from behind forward, and the reverse, so that not rarely the grinding of the teeth can be heard at a considerable distance. The tongue is lashed to and fro in the mouth, not rarely being caught between the teeth and becoming imprisoned there, so that it is bitten, and blood-streaked sputum escapes from the mouth. Generally, frothy saliva escapes from between the lips, probably because in consequence of clonic spasm of the muscles of deglutition the saliva cannot be swallowed. Clonic spasm of the muscles of the trunk gives rise to violent agitation of the entire body, and the patients often throw themselves about from side to side. Respiration is irregular, intermittent, and frequently accompanied by groaning, gurgling, and snoring sounds. The extremities also exhibit the most varied displacements and movements. The fingers are held flexed, and the thumbs drawn beneath the fingers, so that the thumbs can be secured and extended only with difficulty and at times only with luxation or even fracture. The force of the muscular contraction is very considerable, so that occasionally teeth are broken and bones dislocated or fractured. Marked spasm of the muscles of the neck causes stagnation of blood in the jugular veins, so that the external jugular veins often become converted into blue strands as thick as a finger. In addition, there may be present acute protrusion of the eyeballs of progressive intensity, marked conjunctival injection, and cyanosis of the face. Subcutaneous hemorrhages also readily occur. The *pupils*, which were dilated at the commencement of the attack and during the short period of tonic spasm, become contracted with the onset of the clonic muscular spasm. It is of especial diagnostic importance that the pupils exhibit no reaction to light and to sensory irritation of the skin, thus no alteration in size. Often during the attack *involuntary discharge of urine* and also *ejaculation of seminal fluid* take place.

The *duration of an epileptic attack* generally varies from ten seconds to five minutes. The muscular contractions succeed one another with progressively lessening frequency and intensity, cease gradually, and the patient gradually emerges from his coma without knowing what has happened. Some patients are speedily restored to consciousness, and feel fresher and better after the attack than before. Others become fretful, peevish, capricious, irritable, and ill-tempered. In still others hallucinations and illusions appear. They become violent, destroy articles about them, set fire to inflammable articles, attempt to strangle or to kill their attendants—in short, commit crimes without consciousness thereof or without any recollection thereof after emerging from the epileptic attack. These phenomena are grouped together under the designation of *post-epileptic (post-paroxysmal) states.* Further, this condition persists at times for days. Naturally, its recognition is of importance for the comprehension of some otherwise inexplicable crimes.

Epileptic attacks occur at times only by day, in other instances, however, exclusively at night, and, accordingly, a distinction can be made between *diurnal epilepsy* and *nocturnal epilepsy.* Cases of the latter variety are at times surrounded by diagnostic difficulty. Attention should be directed especially to the discovery of whether injury of the tongue, subcutaneous hemorrhage, or involuntary discharge of urine has taken place during the night. The urine at times contains albumin or spermatozoids after an epileptic attack. Occasionally *polyuria*, *glycosuria*, and *paralysis* develop in the sequence of an epileptic attack, the last-named disappearing generally in the course of a few days.

The *number of epileptic attacks* is quite variable. Some patients have perhaps a small number of attacks in the course of a year, while others have an attack or more than one daily. It also frequently happens that periods with few attacks alternate with periods in which numerous attacks occur. Occasionally the attacks occur in such rapid succession that one has not terminated before the other sets in. The patients do not return to consciousness. Such a condition is designated *status epilepticus.* It is not unattended with danger. A rising temperature (up to 42° C.—107.6° F.) particularly is not a favorable sign, and death may be threatened from exhaustion of the central nervous system.

The individual epileptic attack is not rarely followed by *complications. Bite-wounds of the tongue, subcutaneous hemorrhage,* and *albuminuria* have already been mentioned. In the act of falling to the ground a number of more or less serious *injuries* and *cicatricial formations* may result, especially an *othematoma* at the ear. Occasionally an epileptic attack causes *death by asphyxia*, either because of excessive tonic and clonic spasm of the respiratory muscles or because of the occurrence of the attack during a meal, and articles of food gain entrance into the air-passages, or because the patient has fallen awkwardly upon his face, with resulting obstruction of the oral and nasal orifices.

Epilepsy of long standing frequently gives rise to *physical and mental changes*. The patients often acquire a dull, staring expression. The face presents a plump appearance, and memory becomes impaired. The patient gradually becomes demented, and eventually winds up in a hospital for the insane. Often asymmetry of the skull, adhesion of the lobule of the ear, and other so-called *stigmata of degeneration* are present. Naturally highly gifted persons may also be epileptic. Cæsar, Mahommed, and Napoleon furnish examples.

Mild or minor epilepsy appears with especial frequency in the form of *epileptic absences*. The patient suddenly becomes pale, loses consciousness for a few seconds, and the body sways, the eyes are closed or the lids are blinked, and a movement begun is not finished. Consciousness is, however, quickly restored, repeated yawning frequently occurs, the movement is continued, and the patient has no recollection of the entire process. If he has been engaged in conversation, he suddenly ceases during such an attack, and at its conclusion continues with his remarks. Such attacks occasionally occur with great frequency in the course of the day. *Epileptic vertigo* also is a variety of mild epilepsy. The patient is seized with attacks of vertigo, which are, however, nothing more than periods of brief loss of consciousness, although there is generally time to grasp hold of some object and avoid falling. *Tonic and clonic muscular spasm* in a few muscles or extremities, of slight intensity and brief duration, with only indications of disturbance of consciousness, are also comprised in mild epilepsy. Minor epilepsy may retain its mild character throughout life. In some cases, however, it is gradually transformed into severe epilepsy. Naturally, it occurs at times that severe epilepsy is transformed into mild epilepsy.

Epileptoid states, also designated *epileptic equivalents*, exhibit great variations in form, and their nature is often difficult of recognition. Some offences and crimes (breaches of morality, incendiarism, murder) have been committed during epileptoid states. The patient commits such acts while unconscious, and has no recollection thereof. It has been observed that certain patients, moved by a morbid impulse, have undertaken long journeys across the sea, apparently conducting themselves with perfect intelligence, but then suddenly returning to consciousness in a strange country without comprehending how this came about. In some an impulse arises periodically to fall forward, to rotate in a circle —*procursive epilepsy*—movements that may be executed without consciousness. Occasionally such manifestations precede a fully developed epileptic attack. Also, sweating of periodic occurrence —so-called *epileptoid sweats*—*headache*, *attacks of sopor*—so-called *narco-epilepsy*—of *tremor*, *blindness*, *neuralgia*, *hemianesthesia*, and the like, may be of epileptic origin and represent epileptoid states.

Anatomic Alterations and Pathogenesis.—Constant and distinctive anatomic alterations attending true epilepsy are as yet unknown. Attention has been called repeatedly to **atrophy of the cornu ammonis**, and recently **proliferation of the glia in the cerebral cortex** has also been described. In all probability the epileptic attack is the result of *circulatory disturbances* in the motor areas of the cerebral cortex (anterior and posterior central convolutions) and the condition is probably one of hyperemia rather than of anemia within the structures named. In addition to the motor those areas of the cerebral cortex also are involved that maintain consciousness. In favor of involvement of the motor cortical centers is the circumstance that the distribution of the muscular spasm accords entirely with the anatomic relations of the motor cortical centers. If the motor pyramidal tract is interrupted in the vicinity of the internal capsule, clonic muscular spasm will not take place in the paralyzed members. Experimental investigation has shown that epileptiform convulsions can be excited also by irritation of sufficient intensity of other portions of the cerebral cortex. Epileptic states can be induced with especial facility in guinea-pigs, as, for instance, by blows upon the head with a mallet, or after division of the sciatic nerves, and it is noteworthy that the condition may be transmitted to the offspring. In explanation of the occurrence of the epileptic attack the suggestion has recently been made that it may be the result of auto-intoxication from the intestinal tract.

Diagnosis.—The recognition of epilepsy is easy if one has witnessed an epileptic attack, and even the description of such an attack by a layman may generally be utilized for diagnostic purposes, and all the more so if bite-wounds or cicatrices from such wounds upon the tongue or cicatrices upon the skin are demonstrable. Nevertheless, cases occur in which **simulation of an epileptic attack** is practised, particularly upon the part of recruits who wish to evade military service. Under such conditions, however, wounds and cicatrices are wanting, because in falling at the commencement of the simulated attack the malingerer is careful to avoid injury. It is noteworthy that in such an attack the pupils react to light-stimulation and to irritation of the skin. In the simulated attack also the thumb, when abducted and extended, is generally withdrawn into the palm. It is not rarely possible to expose a malingerer by means of various devices. If, for instance, the statement is made before the patient in a loud voice that some symptom not common to epilepsy will occur, the malingerer will often bring about its occurrence; or if warm water is asked for, while actually cold water is brought, and this is poured over the patient, he will, if a malingerer, be taken by surprise, draw back, and disclose the simulation more or less clearly.

Occasionally there may be danger of confounding epilepsy with **uremic** or other **general clonic muscular spasm** (convulsions of dentition or due to worms), and these conditions also have been designated *eclampsia* or *acute epilepsy*. In the first place, however, eclampsia is characterized by but a single attack, or by but a few attacks, and the disorder is not a chronic one; and besides it is attended with other manifestations of organic disease, such as albuminuria, hematuria, hypertrophy of the left side of the heart in cases of acute and chronic interstitial nephritis, irruption of the teeth in association with the eclamptic attacks of infants, and the like. Epilepsy is distinguished from so-called **hystero-epilepsy** by the fact that in the latter the loss of consciousness is generally not complete, that the pupils usually react to light during the attack, that injury is, as a rule, not inflicted during the attack, that the patients generally cry throughout the entire attack, that the attack often lasts for a long time, and that other symptoms of hysteria can be detected with especial frequency—absence of the pharyngeal reflex, contraction of the visual field, and cutaneous anesthesia, particularly hemianesthesia.

It is exceedingly important, in relation to prognosis and treatment, to determine the *cause* of the epilepsy in the individual case, and especially to make a rigid differentiation between true and symptomatic epilepsy. In cases of symptomatic epilepsy consciousness often is preserved during the attack, the convulsions often exhibit a monoplegiform or a unilateral distribution, they always begin in the same extremity, and usually general cerebral manifestations (headache, vertigo, vomiting, slowing of the pulse, papillitis, etc.) are present also in the intervals between the attacks.

Prognosis.—Recovery from true epilepsy occurs but rarely, and can be hoped for only when the causative conditions are of a curable character. Further, care must be exercised in expressing an opinion with regard to recovery, as at times several years elapse before a new attack occurs.

Treatment.—**Causal treatment** will be indicated in the presence of reflex epilepsy, alcoholism, lead-poisoning, and syphilis. Occasionally a cure has been effected by the excision of nerves imprisoned in cicatrices. In **symptomatic treatment** the use of **lcohol** should be interdicted, an unirritating diet prescribed, and the patient advised to engage in a *pursuit* in which he is not exposed to the danger of serious injury or of loss of life in falling. Epileptics should, when possible, be **deterred from marrying**, as, in the first place, sexual intercourse is injurious, and, besides, the disease may be transmitted by heredity. Epileptic mothers should not nurse their children. The victims of severe epilepsy are best taken care of in asylums, such as that admirable one, among others, which has been established through private philanthropy in the canton of Zurich. Of internal remedies, the **bromids** have the highest reputation (sodium bromid, potassium bromid, ammonium bromid, lithium bromid),[1] as, for instance:

℞ Potassium bromid,	50.0 (1½ ounces);
Distilled water, sufficient to make	300.0 (9 fluidounces).—M.

Dose: 15 c.c. (1 tablespoonful) thrice daily.

℞ Sodium bromid,	30.0 (1 ounce);
Potassium bromid,	15.0 (½ ");
Ammonium bromid,	5.0 (75 grains);
Distilled water, sufficient to make	300.0 (9 fluidounces).—M.

Dose: 15 c.c. (1 tablespoonful) thrice daily.

It is preferable to administer bromids after eating and largely diluted. In some patients bromid-acne soon appears, and occasionally extensive furunculosis develops. The remedy must be persisted in for months, and even for years, although then impairment of memory and of sexual power has occasionally been observed. All other remedies that have been recommended for the treatment of epilepsy are, as a rule, not to be compared with the bromids in their effects. It will suffice to mention here *sodium biborate* (50.0 : 300—1½ ounces : 9 fluidounces; 15 c.c.—1 tablespoonful—thrice daily), *belladonna, arsenic, valerian, silver nitrate, opium, morphin, antipyrin, electricity, ligation of the vertebral artery*, and *excision of the superior cervical ganglion of the sympathetic nerve*.[2] Recently *trephining of the skull* has been undertaken for the relief of true epilepsy, but this operation has been discouraged by conservative surgeons.

In order to prevent the occurrence of an epileptic attack, **sodium chlorid** has been swallowed in teaspoonful-amounts, or **one or more extremities have been ligated**, although the patients then not rarely state that they feel much worse than after recovery from an epileptic attack. During the attack care should be taken to prevent the patients from injuring themselves. In the *status epilepticus* long-continued inhalation of chloroform may be recommended.

TETANY.

Etiology.—Tetany is attended with attacks of tonic spasm in certain groups of muscles, the nerves supplying which exhibit increased electric and mechanical irritability. The causative factors include **infectious, autotoxic,** and **toxic influences.** In favor of the infectious nature of some cases of tetany is the circumstance not only that the disease has in several instances been observed to assume an epidemic character in the spring months (March, April), but also that it occurs in the course of or following infectious diseases, as, for instance, pneumonia, typhoid fever, cholera, measles, and small-pox. The condition that follows *complete extirpation of the thyroid gland*, and which has been induced experimentally in animals, is an instance of *tetany of autotoxic origin.* It has been possible to prevent the development of the condition in animals by transplantation of thyroid tissue in the abdominal cavity. It is believed that the thyroid gland secretes a mucoid material whose retention in the blood exerts a toxic influence upon the central nervous system. Under the same heading probably belongs also the tetany that occurs in the course of *exophthalmic goiter.* Occasionally tetany develops in connection with *chronic gastric and intestinal disease*, and with especial frequency in connection with dilatation of the stomach and helminthiasis. Tetany has also been not rarely observed in cases of *nephritis, pregnancy,* and *lactation. Tetany of toxic origin* develops after excessive indulgence in alcohol, after inhalation of ether and of chloroform, and in cases of lead-poisoning.

---

[1] When the pulse is rapid and the vascular tension is low the conjoint admin-

While tetany occurs quite frequently in certain *regions* (Vienna, Prague), it is a most uncommon disorder in other places (Berlin, Zurich), although no cause has as yet been discovered for the difference. The disease is frequent in *children* between the first and the third year of life. It occurs next in frequency between the thirteenth and the thirtieth year of life, while it is uncommon after the fortieth year. *Pallid and rachitic children*, as well as individuals in families with an hereditary neurotic predisposition, are especially prone to be attacked by the disease. It is a remarkable fact that the disease is particularly common among those engaged in certain **occupations,** particularly shoemakers and tailors, as well as cabinet-makers and locksmiths. **Exposure to cold, emotional disturbances,** and **peripheral irritation** (irruption of the teeth, over-distention of the stomach and intestines, irrigation of the stomach, menstruation) also appear to favor the occurrence of an attack of tetany.

Symptoms and Diagnosis.—An attack of tetany is frequently preceded by *prodromes*, consisting in part in paresthesia in the extremities that subsequently become the seat of spasm, and in part of cerebral disturbances (mental confusion, vertigo, headache). The *attack of tetany* involves in typical cases the **flexors of the hands and the fingers.** The thumb is held beneath the flexed fingers; the hand is in a position of ulnar flexion, and excavated as for the purpose of scooping up water; the fingers and the hand are suggestive of those of the obstetrician in the act of introducing the member into the vagina (obstetric hand). The hand may also assume the attitude taken in writing. As a rule, the hand and the fingers on each side are involved in the tonic spasm, which can be temporarily overcome by some force, but which recurs at once as soon as the traction upon the muscles is suspended. Often the flexors of the forearm also participate in the tonic spasm, so that the forearm is held flexed at an acute angle with the upper arm. The adductors of the upper arm also are not rarely involved in tonic spasm, with resulting approximation of the upper arm to the chest, and even to crossing of the elbows. The muscles, in a state of permanent contraction, are hard, and their outlines are distinctly visible beneath the skin. Pressure upon the contracted muscles generally excites pain, and the patients often complain spontaneously of painful contractions and paresthesiæ of varied kind.

Although attacks of tetany involve most frequently the muscles of the hand, the fingers, and the arm, similar tonic spasm occurs by no means rarely in addition, or alone, in **other groups of muscles.** In the lower extremities the flexors of the toes are involved with especial frequency, and under such conditions the strongly flexed great toe is drawn beneath the adjacent toes. Likewise, the flexors of the foot, but occasionally also the extensors, may be involved in the spasm. In the thighs tonic contraction of the adductors and the extensors occurs not rarely. The **muscles of the trunk** also are at times involved in tonic spasm. The abdominal walls are contracted with board-like hardness; or in consequence of spasm of the thoracic muscles, and even of the diaphragm, respiratory disturbances occur, and tonic spasm of the muscles of the back causes distortion of the vertebral column or of the muscles of the neck—rigidity of the neck. The **muscles of the face and of mastication** may also be involved. Even the muscles of the eye, the tongue, the pharynx, the esophagus, the larynx, and the urinary bladder have at times been involved.

The *duration of an attack of tetany* varies between a few seconds and several days. The *recurrence of the attacks* is likewise susceptible of great variation. In some patients but a single attack occurs, while in others numerous attacks occur at short intervals in the course of a day. The **tendon-reflexes** in the group of muscles involved exhibit no constant alteration, as they have been variously found unchanged, enfeebled, and increased. The **bodily temperature** is generally unchanged at the time of the attack, although an elevation has also been described. **Consciousness,** likewise, is, as a rule, unaltered. Occasionally, it is true, psychopathic states develop.

At times *edema, redness*, and *swelling of the joints, cyanosis, pigmentation of the skin*, and *herpes* have been observed. *Pressure-points* also are not rarely mentioned as being present along the *vertebral column.*

Of especial importance in the diagnosis is the observation that the **electric and mechanical irritability of the nerves** supplying the muscles involved in the spasm is increased. Similar alterations have been found also in the sensory nerves. Of equal importance in the recognition of the disorder is the presence of the **Trousseau phenomenon.** If, in the interval between attacks, the arm or the leg respectively is constricted for one or two minutes, or if pressure is made with the fingers upon the large nerve-trunks or upon the adjacent arteries, it is possible to excite an attack of spasm. This sign becomes the more important, as it renders possible the recognition of latent tetany, for as long as it is demonstrable the disease cannot be considered as cured, and there will be danger that new attacks will occur spontaneously. Less importance is to be attached to the **facial phenomenon** first described by Chvostek, which consists in active contractions of the facial muscles when a hard body, as, for instance, the handle of a plexor, is drawn transversely over the facial nerve. This phenomenon occurs also independently of tetany in nervous and anemic persons.

The *duration of tetany* is variable, and a distinction is made between *acute*, *subacute*, and *chronic tetany*, accordingly as the disorder persists for two or six weeks, or for many years (up to twenty). In the event last named remissions and exacerbations frequently alternate. There is a great tendency to *recurrence*. Such a condition is observed in women with each pregnancy. *Muscular contractures*, *paralysis*, and *epilepsy* have been observed as *sequelæ*. *Atypical* or *masked tetany* is, further, worthy of consideration. In this must be included the *tetanoid states*, in which, while the electric and mechanical irritability of the nerves is increased, and Trousseau's and Chvostek's phenomena are demonstrable, muscular spasm does not occur spontaneously. Nevertheless, tetanoid states may be transformed into well-developed tetany. Some clinicians consider *laryngismus stridulus* or *spasm of the glottis* as a variety of atypical tetany.

Prognosis.—Tetany is, in general, a benign disorder. A fatal result has been observed frequently only in cases of tetanus secondary to dilatation of the stomach.

Treatment.—The treatment of tetany should, in the first place, be **causal.** Occasionally, rapid recovery has been observed to take place after the administration of an anthelmintic. Nursing women should at once remove the child from the breast. In cases of dilatation of the stomach an effort should be made to remove toxic substances from the stomach by means of irrigation, although care is required in this procedure, as muscular spasm has been observed to follow irrigation of the stomach. Complete removal of the thyroid gland should be avoided also for the purpose of preventing the development of myxedema, and in the absence or deficiency of the thyroid gland thyroid medication should be instituted. Under all circumstances the patients should secure full bodily and mental rest, partake of a non-stimulating **diet**, avoiding particularly alcohol and strong tea and coffee, and secure a regular **evacuation of the bowels.** Among **medicaments**, the various *nervines* and *narcotics* have been employed, although no especially striking results can be attributed to any one of them. We prefer the *bromids*, believing the disease to be due to undue irritability of the ganglion-cells in the motor cortical areas of the brain, and that the bromids reduce this irritability. **Electric treatment** of the diseased muscles and nerves, of the spinal cord, the brain, and even of the sympathetic nerve has also been practised.

OCCUPATION-NEUROSES.

Occupation-neuroses are characterized by loss of orderly and coördinated activity on the part of groups of muscles that should coöperate with precision in the execution of definite movements, with resulting more or less marked difficulty in the performance of the given movements. *Writers' cramp* is the form of occupation-neurosis that has been longest known, and that occurs most frequently, and for this reason it will be considered rather in detail.

WRITERS' CRAMP (MOGIGRAPHIA; SCRIVENERS' PALSY).

Etiology.—Writers' cramp occurs, as a rule, in *adults*, and generally in *men*. In the majority of cases a **neurotic tendency** can be demonstrated, which is either inherited or acquired through an injudicious mode of life or emotional disturbances. Only rarely are persons previously in perfect health attacked by writers' spasm. This condition may result from excessive writing, or the use of unsuitable pen-holders and pens, or of improper surfaces for writing, the wearing of cuffs that are too tight, the pressure exerted by cuff-buttons, from a blow, and from inflammation of the brachial nerves or the periosteal covering of the bones of the arm.

Symptoms and Diagnosis.—The symptoms of writers' cramp are frequently preceded by **prodromes**, such as mental confusion, a sense of pressure in the head, vertigo, irritability, a sense of fear, headache, and the like. Four varieties of writers' cramp are generally described, and these are designated spastic, tremulous, paralytic, and neuralgic.

The *spastic variety of writers' cramp* is the most common. On attempts at writing, particularly after writing for a long time, tonic or clonic spasm occurs in the muscles employed in this act, so that it is in consequence interfered with or prevented. Flexor or extensor spasm of the muscles of the thumb prevents proper guidance of the pen, as does also flexor spasm in the index-finger and the middle finger; while the pen cannot be held in the presence of extensor spasm of the muscles named. Spasm of the muscles of the little finger causes marked difficulty in writing. The muscles of the forearm also often participate in the spasm, in consequence of which sudden contraction of the ulnar flexor of the carpus may cause the pen and the hand to tear through the paper, and the hand may be jerked away in writing. Even the muscles of the upper arm, the shoulder, and the neck, and at times also muscles still further removed may be involved in the spasm. The patient often complains during the spasm of disagreeable and even painful sensations and **paresthesiæ** in the affected parts, and in many cases a feeling of fear is present, with sweating and palpitation of the heart. The muscles and some of the nerves of the arm are at times markedly sensitive on pressure. **Painful points** also occur upon the bones of the arm, particularly at their articular extremities, and over the spinous processes of the vertebræ. The symptoms are generally aggravated when the patients believe that they are observed or when they take especial pains to write beautifully and with steadiness. The progress of the disease can best be followed from the character of the writing, which gradually comes to resemble illegible scrawling. Often the adoption of a new penholder, or of a new pen, or a new writing-surface, or another mode of holding the pen, writing with the right hand grasped by the left, brings temporary relief, but even each of these devices fails after a while, and writing becomes impossible.

In the *tremulous variety of writers' cramp* tremulous movements occur as soon as the patient undertakes to write. The tremor increases with the advance of the disease, and the writing accordingly becomes progressively more illegible. *Neuralgic writers' cramp* is attended with intense pain in the act of writing, and *paralytic writers' cramp* with a feeling of marked weakness in the muscles employed in the act of writing, so that the hand rests helplessly upon the paper and cannot be moved.

Prognosis.—Writers' cramp is unattended with danger to life, but it is a most obstinate and generally incurable disorder, and it is of the greater significance as it occurs, as a rule, in persons who are compelled to write a good deal and continuously.

Treatment.—The treatment may be divided into **general** and **local**. The *general treatment* should be directed to the relief of the neurotic state, and to this end particularly *nervines*, and especially bromids, in anemic cases *preparations of iron*, further courses of treatment with *cold water*, and *climatic treatment in the mountains or at the seaside* should be employed. The patient should abstain from his work for as long a time as possible, and receive a nutritious, unstimulating *diet*, from which alcoholics especially should be excluded. Among the most important *local remedies* is long-continued *abstinence from all attempts at writing.* Many patients acquire the ability to write with the left hand, but after a time the same disorder makes its appearance also in that member. If it be possible to find pressure-points over the bones of the arm or over the nerves, these should be treated with narcotic ointments or with anodal applications of the galvanic current. Good results are frequently obtained from **massage.** The employment of a **thick penholder** and of a **finger-support** for the pen is a makeshift that is helpful for only a short time. Occasionally relief has been afforded through **galvanization,** and, according to reports, also through **hypnosis.**

Among other occupation-neuroses, the following may be mentioned: Piano-players', organists', violinists', zither-players', harpists', telegraphers', tailors', shoemakers', smiths', masons', cloth-fullers', sawyers', weavers', watchmakers', engravers', draftsmen's, compositors', cigaret-makers', knitters', cashiers,' and milkers' spasm. Occupation-neuroses involving the muscles of the lower extremities have been observed in those who work organ-bellows with their feet, in turners, sewing-machine operators, and dancers. Similar conditions have been described as occurring also in the muscles of the lips and the tongue in flutists, trumpeters, and clarionetists. The character of the disturbances resembles that of those described for writers' spasm.[1]

CHOREA (ST. VITUS' DANCE).

Etiology.—St. Vitus' dance, also designated *chorea minor* or *Sydenham's chorea,* is a *frequent disease of childhood,* which usually occurs at about the period of the second dentition (sixth or seventh year) and at puberty (from the eleventh to the fifteenth year), and in *girls* with almost twice the frequency as in boys. Adults are attacked much less commonly. In rare instances the disorder appears in old age—*senile chorea.* Chorea occurs with especial frequency in such children as are the offspring of neurotic families or as become neurotic through improper modes of living, particularly through over-exertion at school and too close confinement indoors. In all probability chorea is dependent upon disturbances in the motor and psychic areas of the cerebral cortex, in consequence of **infection, reflex irritation,** or **psychic influences,** and possibly also of **toxic** and **autotoxic influences.**

The *infectious varieties of chorea* include the cases in which the disorder appears as an *independent infectious disease* often in epidemic distribution or in the course or as a sequel of infectious diseases. It has frequently been observed that epidemics of chorea occur among children in some localities, particularly in the months from February to April, although it must be admitted that the infectious agent has not yet been isolated. Of the infectious diseases in connection with which chorea occurs not infrequently is especially *acute articular rheumatism*. In adults *syphilis* also is to be taken into consideration.

---

[1] A disorder of like character has been observed in typewriters, treadlers, motormen. and others.—A. A. E.

Reflex chorea may originate from any of the viscera. In children it has been observed in connection with caries of the teeth, intestinal worms, and coprostasis; the chorea rapidly subsides after correction of the conditions named. The *chorea of pregnancy*, which generally occurs in primigravida in the first months of pregnancy, is probably only a variety of reflex chorea. Not rarely chorea occurs in association with *valvular disease of the heart*, and under such conditions it is not inconceivable that the hypertrophied heart may exert an irritating influence upon adjacent nerves, as, for instance, the phrenic, as it passes in front of the pericardium, and thus may excite an attack of chorea through reflex action. The *psychic influence* in the development of chorea can be recognized from the fact that some children are attacked by the disease immediately after a severe *fright*.

In addition, the importance of *imitation* is worthy of mention. Experience has shown that occasionally children are attacked by chorea who have associated with others suffering from this disease. Chorea is, therefore, sometimes observed to occur in endemic distribution in institutions (schools, orphan-asylums, boarding-schools), although confusion with similar hysterical states must be guarded against. *Toxic chorea* has been observed after the use of iodoform and in connection with mercurialism. Perhaps an *autotoxic variety of chorea* should be added to those already described. Thus, the disease has been observed to occur in cases of progressive pernicious anemia and of nephritis.

Symptoms.—The onset of an attack of chorea is not rarely preceded for a few days or weeks by **prodromes**, consisting in change in character, capriciousness, irritability, timidity, restless sleep disturbed by dreams, impaired appetite, increasing pallor, and similar manifestations. The distinctive features of chorea consist in **muscular restlessness, incoordinate movements**, and **alteration in the mental state**. As a rule, the disorder of movement *begins gradually*. In dressing, eating, writing, and in various other manipulations the patients exhibit unusual awkwardness, and the attendant is generally disposed to attribute this to carelessness or even to viciousness. Soon, however, the motor disturbance becomes so pronounced that often even a layman is capable of diagnosing the well-known disorder.

In the first place, the *muscular restlessness* is conspicuous, preventing the patient from sitting, standing, or lying still for any length of time. At times there appear in the face, at other times in the fingers and the arm, then again in the toes and the feet, muscular contractions and movements that occur wholly without purpose. In severe cases the patients are unable to keep still for the shortest possible time, throwing themselves about in bed, raising the body, at times jerking the arms and legs forward and again backward, rotating the head from side to side and retracting it upon the nape of the neck, then approximating the chin to the chest—in brief, the entire muscular system is involved in uninterrupted, at times less marked, at other times more marked, movement. Often high protecting boards must be placed at the side of the bed in order to prevent the patient from falling out. As a rule, the patient makes no complaint of fatigue or of pain, even though unable to remain quiet for even a few seconds and although the movements are extremely vigorous.

The *character of the muscular movements* is quite striking. All of the movements exhibit clumsiness and awkwardness, and appear sprawling and exaggerated. No movement is executed in the most direct and the shortest manner, but each is interrupted and disturbed by jerky and secondary movements. It can be readily seen that these peculiarities result from the fact that the normal character and course of voluntary movements are interfered with by involuntary and incoördinate muscular contractions. Naturally, muscular restlessness and incoördinate muscular movements give rise to marked disturbances. The gait of the patient not rarely takes the form of active jumping, whence the name chorea, or St. Vitus' dance, may be readily understood. At other times the gait resembles the movements of a skater, and is attended with zigzag movements. It may, however, also happen that the patient is wholly unable to walk, the feet becoming entangled in attempts at walking, so that the patient falls. In the arms movements of pronation, supination, extension, and flexion succeed one another with irregular alternation. The patients often are unable to carry food to the mouth, and must be fed; they are unable to dress themselves, to button their clothing, to sew, or to write. Particularly delicate manipulations, as, for instance, the threading of a needle or the passage of a needle through a previously made perforation, are executed with especial difficulty.

Should the *muscles of the trunk* also be involved in the choreic movements, interruption and irregularity in the respiratory movements, or jerky retraction of the abdominal wall, may be observed from time to time. The *face* exhibits involuntary grimaces, such as sudden wrinkling of the forehead or the eyebrows, pursing of the mouth in alternation with widening of the oral fissure, snuffling movements with the alæ of the nose, or contortions of all of the facial muscles. The *tongue* also is not rarely involved. When protruded, it makes movements to and fro, and may then be suddenly jerked back into the mouth. The *ocular muscles*, however, almost always remain uninvolved. On the other hand, the *muscles of the soft palate* and of the *vocal bands* take part in the chorea, and often marked *articulatory disturbances* are noticeable, so that speech is scanning or wholly unintelligible. The *heart-muscle* continues its contractions regularly even when the disease is widespread and marked.

Almost unexceptionally the **mental condition** of the patient is affected. The patient becomes peevish, foolish, lacrimose, disconcerted; but marked psychopathic states, such as acute mania, also occur, and these may render the prognosis serious.

All choreic movements have a tendency to be increased under the influence of observation and embarrassment. They may be temporarily moderated, and even suppressed, by an effort of the will. They cease during sleep, but appear during dreams. Often the patient is able to fall asleep only with difficulty.

Only that variety of chorea has thus far been considered in which the movements involve the entire body and which may therefore be designated *total* or *general chorea.* In contradistinction from this is *partial chorea,* in which the choreic movements are confined to one member—*monoplegiform chorea;* or both arms or both legs are involved—*superior* or *inferior paraplegiform chorea;* or the arm and the leg upon the same side, or upon opposite sides, are involved—*hemichorea, chorea dimidiata,* or *alternating chorea.* Frequently chorea begins as the partial variety, and is gradually transformed into general chorea.

Chorea pursues an *afebrile course.* Elevation of the bodily temperature is an unfavorable sign, particularly if it attains hyperpyretic figures (above 42° C.—107.6° F.). The patients frequently become pallid with exceeding rapidity, and the right ventricle of the heart often undergoes anemic dilatation, with the appearance of cardiac-systolic **anemic murmurs,** so that confusion with valvular disease of the heart must be guarded against, as this condition occurs by no means rarely in cases of chorea—as has already been pointed out. Occasionally, **pressure-points** and **painful points** can be demonstrated over the vertebral column, or at the articular ends of the bones of the extremities. Peripheral nerve-trunks also at times exhibit pressure-points or painful points on application of the electrode of a galvanic current. Some patients present **painful swelling of the joints,** during the presence of which the choreic movements occasionally subside. **Nodose rheumatism** has often been observed, and in connection with which nodules form particularly in the tendons. At times marked **hysterical disturbances** occur, as, for instance, hysterical mutism. **Paralysis of the bladder** and **atrophy and paralysis of one or more of the extremities** may develop.

Chorea generally pursues a *chronic course,* and lasts from four to twelve weeks. It disappears quite gradually, the choreic movements becoming progressively slighter. There is a great tendency to *recurrence.* In women recurrence has not rarely been observed with each successive pregnancy. Often the recurrent attacks are much more marked and more protracted than the original attack.

Diagnosis.—Chorea can scarcely be mistaken for any other disease on account of its readily recognized and peculiar disorder of movement. Occasionally, choreiform movements appear in association with diseases of the brain (meningitis, softening, neoplasm)—so-called *symptomatic chorea;* but other cerebral manifestations will likewise be present. **Prehemiplegic and posthemiplegic chorea** are attended with choreiform movements only in the hemiplegic members. At times choreiform movements are observed in **hysterical patients** (chorea major), although other symptoms of hysteria will also be present. In the presence of **athetosis** the movements are sprawling rather than incoördinate, they occur particularly in the fingers and the toes, and the affected parts are paralyzed. **Shaking palsy** or **paralysis agitans** is a disease of old persons; the movements resemble those of marked tremor, are unattended with incoördination, and continue uninterruptedly. The **intention-tremor attending multiple cerebrospinal sclerosis,** finally, appears only on voluntary movement.

Prognosis.—The prognosis of chorea is not unfavorable, and particularly in children death occurs but rarely. It is also uncommon for the disease to prove incurable and to persist throughout life. Maniacal states must be considered as an unfavorable indication.

Treatment.—**Causal treatment** is applicable especially in cases of reflex chorea, and will vary with the etiologic factors in the individual case. In cases of infectious chorea salicylic acid, sodium salicylate, antipyrin, or phenacetin may be prescribed, and favorable reports of the action of these remedies have been made, although I have personally observed no brilliant results from their use. Under all circumstances an intelligent **dietetic regimen** should be observed. The patient should remain in bed, and receive a diet in which milk preponderates. The use of coffee, tea, and strong alcoholics should be forbidden. The patient should be cautioned to refrain from all mental activity, from reading, from listening to stories, and from the use of picture-books. An admonition to suppress the movements as largely as possible may properly be made. Daily evacuation of the bowels should not be neglected. **Warm baths** at a temperature of 35° C. (28° R.—95° F.), and of a duration of from fifteen to thirty minutes, may be employed with advantage. To me it appears a matter of indifference whether 100.0 (3 ounces) of potassium sulphid are added to the bath or not. Among internal remedies, **nervines** and **narcotics** have been recommended, although care should be observed in the use of the latter, as this is not rarely followed by alarming collapse. Personally, I prescribe either **arsenic** or **bromids,** and rather prefer

℞ Solution of potassium arsenite,
Bitter-almond water, each, 5.0 (75 minims).—M.
Dose: 5 drops thrice daily after eating, and increasing 1 drop every three days, until 10 drops are taken thrice daily.

℞ Solution of sodium
bromid, 10.0 : 200 (2½ drams : 6½ fluidounces);
Potassium bromid, 5.0 (75 grains);
Ammonium bromid, 3.0 (45 ").—M.
Dose: 5 c.c. (1 teaspoonful) thrice daily after eating.

HEREDITARY CHOREA OF ADULTS.

Hereditary chorea of adults was first fully described by Huntington, and is therefore known as *Huntington's chorea*. It is an hereditary disorder in some families, of which it may attack every member; but if one escapes, the hereditary transmission for his posterity is interrupted. The first symptoms of the disorder appear between the *thirtieth and fortieth years of life*, and occur in the muscles of the face, to extend thence to the upper extremities, the trunk, and, finally, also to the legs. The choreic movements are often quite active, and they may involve also the muscles of the tongue and the larynx, and, in consequence, give rise to disturbances in respiration and articulation. It is noteworthy that the patients are able voluntarily to suppress the movements for a time. After a few years the disorder of movement diminishes almost to the point of disappearance, while indications of *mental derangement* become gradually more pronounced. The patients fall into a state of progressive dementia, and often attempt suicide. Amid progressive exhaustion death occurs. Anatomically, *foci of encephalitis* in the cerebral cortex have recently been found. As compared with Sydenham's chorea, Huntington's chorea is distinguished by the heredity, the occurrence in advanced life, the development of mental derangement, and, possibly, also the anatomic findings. The *prognosis* is unfavorable, as *treatment* is powerless.

PREHEMIPLEGIC CHOREA AND POSTHEMIPLEGIC CHOREA.

Occasionally the occurrence of cerebral hemorrhage is preceded by choreic movements in the arm and leg that are subsequently paralyzed in consequence of the hemorrhage—*prehemiplegic chorea;* or the movements appear in the hemiplegic members as soon as these resume their activity—*posthemiplegic chorea*. The degree of disturbance is variable, and the condition is at times recognizable only on careful examination. In cases of prehemiplegic chorea the movements cease with the development of the paralysis, while posthemiplegic chorea occasionally persists indefinitely. Experience has shown that *hemianesthesia* exists in addition to hemiplegia and hemichorea, so that the condition appears to occur especially in connection with destruction in the posterior portion of the internal capsule. Lesions in the pons or in other portions of the motor pyramidal tract may also give rise to the symptoms described.

All **treatment** is inefficacious.

ATHETOSIS.

There are two varieties of athetosis, the symptomatic and the idiopathic.

Symptomatic athetosis is the more common, and occurs in the course of a number of *diseases of the brain and the spinal cord.* It not rarely develops in association with the *cerebral paralysis of childhood*, but it is encountered also in connection with *epilepsy, idiocy, tabes dorsalis*, and *spinal paralysis of childhood. Idiopathic athetosis* has been observed in children whose parents were *drunkards* or had suffered from *syphilis. Emotional disturbances during pregnancy* also are believed to cause athetosis in children. Occasionally *inherited athetosis* has been observed. *Exposure to cold, traumatism, antecedent infectious diseases*, and *emotional disturb-*

and in the cervical cord, although these may possibly have been only accidental alterations.

Cure can scarcely be anticipated, so that the *prognosis* is unfavorable. *Nervines, narcotics, electricity, courses of treatment with cold water, massage,* and *hypnosis* have been employed without successful results.

MYOCLONUS.

The designation myoclonus has been applied to *attacks of clonic muscular contraction* occurring as the result of various external influences, so that four principal varieties have accordingly been distinguished, and these have been designated electric chorea, multiple paramyoclonus, saltatory spasm, and spasmodic tic. This subject is worthy of careful clinical investigation, and transitions from one to another variety occur not rarely.

Electric chorea presents a variable clinical picture. The designation *Dubini's electric chorea* has been applied to a disease observed in Lombardy, which commenced with attacks of clonic muscular spasm in one arm, extending thence to the leg of the same side, and finally involving all four extremities. The muscles wasted, and lost their irritability to the faradic current. Amid progressive exhaustion death generally resulted. The disorder made the impression of an infectious disease.

Bergeron's electric chorea generally occurs in children between seven and fourteen years old. It begins in the muscles of the neck, in which clonic spasm of such severity occurs that the impression is made as if the muscles were stimulated with a strong faradic current. The muscles of the shoulder, the arm, and the trunk are next involved. The disease terminates in a few days in recovery. The patients have generally been pale and nervous children. Fright and gastric disturbances have been mentioned as *causes for the disorder.* The *treatment* consists in regulation of the diet, the administration of arsenic, and the application of a cold douche.

Multiple paramyoclonus is attended with attacks of clonic muscular spasm in symmetric muscles. Occasionally the spasm extends to all of the muscles of the body. The electric and mechanical irritability of the nerves and muscles is unchanged, although exaggeration of the tendon-reflexes has been observed. Antecedent infectious diseases and fright have been mentioned as *causative factors,* although cases have also been observed in which hereditary and familial influences were present. The disorder occasionally persists for several years, and is attended with exacerbations and improvement. Recovery has been brought about in a number of cases by *galvanization of the spinal cord or of painful points over the vertebral column.*

Saltatory spasm has been observed in but a few instances. As soon as the patient has placed his feet upon the ground for the purpose of standing or walking clonic muscular spasm appears, giving rise to dancing and jumping movements of the body. The muscles of the arms, the shoulders, and the nucha also have occasionally been involved in the spasm. The disease has at times resisted all treatment, including baths, the application of ice to the vertebral column, electricity, nervines, and narcotics.

Spasmodic tic generally sets in with *clonic spasm* of the muscles of the face, gradually extending to the muscles of the neck and the nucha, particularly the sternomastoid and the trapezius, and finally the muscles of the extremities. The patients are frequently able to suppress the muscular spasm for a while. After a time *disorders of speech* appear. The patients make sniffling sounds, imitate the cries of animals, constantly repeat the same word that has been spoken by another (echolalia), or blurt out vulgar or offensive words or phrases (coprolalia). In addition, there may be imitation of certain gestures—*echokinesis*. The disease may eventually terminate with *mental derangement*. The disorder often begins in childhood, and may persist throughout the whole of life. The patients generally are neurotic persons. Emotional disturbances, imitation, traumatism, and infectious diseases may be the immediate causes of the disease. The disorder is differentiated from hysteria by the absence of hysteric stigmata. Recovery occurs but rarely. *Nervines, narcotics, electricity, courses of treatment with cold water, gymnastic exercises,* and *setons* have been employed.

PARALYSIS AGITANS (SHAKING PALSY).

Etiology.—Paralysis agitans is a *disease of advanced life*, which generally appears after the fortieth year, and occurs in both sexes with about equal frequency. Among the causative factors especially **emotional disturbances** and **traumatism** may be mentioned. The disease has been observed to develop also in the sequence of **exposure to cold** and **infectious diseases**. In one instance I observed **tabes dorsalis** complicated by paralysis agitans. Recently emphasis has been laid on the *heredity* of the disease.

Symptoms and Diagnosis.—The disease is not rarely preceded by **prodromes**, particularly vertigo, mental confusion, occasionally attacks of vomiting in the manner of gastric crises. The *distinctive symptoms* consist in constant shaking movements, progressive muscular weakness, muscular rigidity, a peculiar attitude of the body, a mask-like expression of the face, and forced movements. The **shaking movement** often appears as a sort of fine tremor, gradually, however, attaining such intensity that the affected parts are thrown to and fro and the whole body is involved in violent shaking. These movements can be temporarily checked by an effort of the will, so that they can be readily differentiated from the intention-tremor of multiple sclerosis, which occurs only on intended movement.[1] Often the shaking movements can be controlled temporarily by grasping the affected members firmly with the hands, ligating them or placing heavy objects upon them, although the patients are made uncomfortable by such procedures, become conscious of a constricting, oppressive feeling, and beg to be quickly released. The number of oscillating movements is four or five in the second. Generally the movements are particularly well developed in the fingers. The digits move up and down, and the tips of the middle finger and the index-finger are approximated to the thumb. Often the tips of the digits mentioned are constantly rubbed upon one another, and the patient presents an appearance as if he were continuously rolling a globule between his fingers or as if he were pulling wool. The movements generally cease during sleep, although the patients often are prevented from falling asleep. Naturally all manipulations on the part of the patients are greatly interfered with by the shaking movements, particularly in dressing, eating, and writing, and often they require assistance for years.

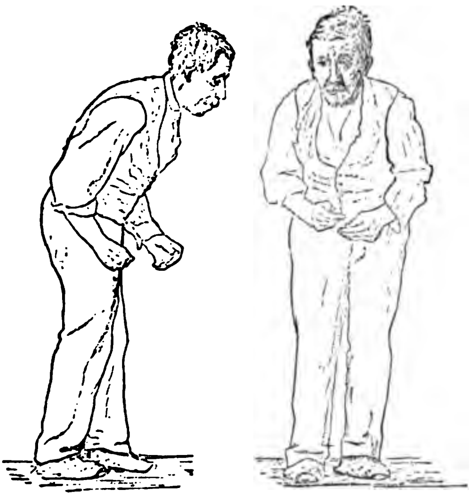


FIG. 17.—Paralysis agitans (after St. Leger).

The tremulous movements described generally make their appearance earliest in one extremity, and the right arm is oftenest first involved. They then extend to the right leg or to the opposite arm, and finally all four members are involved. In any event, the disease may at certain periods of its development exhibit a monoplegiform, a hemiplegiform, or a paraplegiform distribution. Also, the muscles of the head and the nucha, those of the face and of mastication, even those of the tongue, the soft palate, and the vocal bands, rarely the ocular muscles, are occasionally involved in the tremulous movements.

---

[1] Rarely the characteristic tremor is wanting, and it has been stated that at times it is increased by voluntary effort.—A. A. E.

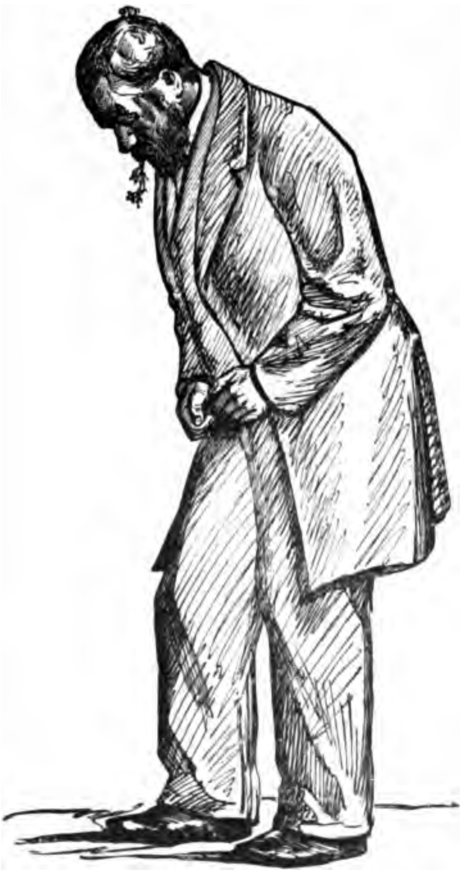


FIG. 18.—Position of the body in a case of paralysis agitans in a man 53 years old; from a photograph (personal observation, Zurich clinic). (The patient held a flower constantly between his teeth in order to keep the jaw still.)

The longer the movements have existed the more marked becomes progressive **weakness of the affected muscles.** The grasp of the hand thus becomes exceedingly feeble. Progressive weakness in the legs requires the patient to occupy the sitting posture a good deal, and gradually makes him permanently bedridden. **Rigidity of the muscles** is readily demonstrable. If the patient be requested quickly to extend and flex the arms alternately he will execute the desired movements but slowly, and generally but incompletely. Passive movement also is attended with distinct resistance. **Muscular contractures** gradually result from this muscular rigidity. The **mask-like appearance of the face** is probably dependent upon the muscular rigidity. The facial expression appears rigid, unchangeable, and grave, and all the more so as speech is monotonous, the voice low and almost wooden in tone.

The extremities and the trunk generally assume a peculiar **attitude.** The neck and the head are bent forward in standing and walking, the upper arms are held off from the chest; the forearms are flexed approximately at a right angle; the hands are generally held in front of the body, and grasp each other in order to prevent the shaking movements (p. 110, Fig. 18). The fingers assume a peculiar position that has been designated the "pen-holder" position. Occasionally, however, the position of the fingers is suggestive of deforming arthritis. The lower extremities are flexed at the hip and the knee, and in walking the patient often experiences difficulty in moving the thighs in front of each other if the adductors are contractured. In walking the patient makes short, stumbling steps, and gives the impression as if he would fall forward. At times, in my own experience but seldom, manifestations of **propulsion, retropulsion,** and **lateropulsion** appear. Propulsion is attended with progressively increased celerity of movement in walking rapidly until the patient falls. Retropulsion and lateropulsion are attended with the danger that the patient, if pushed backward or laterally, makes steps of progressively increasing rapidity until he falls to the ground. It is also noteworthy that the patient is unable immediately to obey the command to halt, but makes a few additional steps. Also in reading, similar disturbances occasionally become apparent, the patient requiring a certain amount of time after reading one line to reach with his eyes the beginning of the succeeding line.

The manifestations of paralysis agitans may be confined to the symptoms that have been described. At any rate, they are distinctive in diagnosis. In rare instances the shaking movements are wanting, while muscular weakness, rigidity, and alteration in facial expression are present. Many patients complain of a **sense of internal heat and restlessness,** and prefer to remain in the neighborhood of an open window. A tendency to **profuse sweating** also is often present. The **bodily temperature** generally remains unchanged. The duration of the disease often extends over many years. Bodily wasting becomes more and more marked, and the patient is often compelled to spend the latter part of his life permanently in bed. Occasionally **apoplectiform seizures** or **states of profound apathy and unconsciousness** occur, which disappear within a short time without leaving permanent disturbances. Death occurs amid progressive exhaustion, occasionally in consequence of cerebral hemorrhage, of bed-sores and septicemia, or of any intercurrent disease.

Prognosis.—The disease is incurable, but it is unattended with immediate danger to life.

Treatment.—No successful treatment of paralysis agitans is as yet known, and a suitable **dietetic regimen** therefore plays the most important *rôle*. The shaking movements may occasionally be mitigated, or even controlled, by means of **narcotics** (opium, morphin, chloral hydrate, trional, sulfonal); but frequently alarming conditions of somnolence, coma, and serious exhaustion occur. Among **nervines** solution of potassium arsenite and bromids may especially be mentioned, although I have observed similar complications to arise after the administration of bromids as after the use of narcotics.

TREMOR.

Symptoms and Diagnosis.—Tremor consists in a constant to-and-fro movement of the affected parts, the intensity, uniformity, and rapidity of the movements being susceptible of great variation. At times the trembling involves a single member or a number of small parts of the body, while at other times it extends to the entire body. The movements can be more or less completely controlled by an effort of the will. They often cease during rest, while they are generally increased by emotional disturbances and by physical exertion. Tremor is in itself not a dangerous condition, although it prevents many persons from the pursuit of their occupations, as a quiet, steady hand is required.

Etiology.—Tremor is often the result of **excessive physical activity,** occurring, for instance, after the carrying of heavy articles. **Emotional disturbances** (joy, fright, fear) occasionally cause tremor. It is often a **symptom of exhaustion.** It is, therefore, observed after severe diseases and in old age—senile tremor. **Toxic tremor** occurs after excessive indulgence in alcohol, tobacco, tea, opium, and morphin, and after poisoning with lead, mercury, and arsenic. **Thermic influences** are operative in the production of tremor by cold. Tremor often occurs in the course of certain **central neuroses,** as, for instance, hysteria, neurasthenia, epilepsy, and exophthalmic goiter. At times it has been observed to develop as an independent disorder through *heredity,* and to begin as early as childhood.

The **prognosis** and the **treatment** depend upon the causative factors. Of drugs, **solution of potassium arsenite, hyoscyamin,** and **zinc phosphid** especially have been recommended.

VERTIGO.

Etiology.—Vertigo is a frequent symptom of **disease of the brain and the spinal cord,** of which it will suffice to name as instances meningitis, cerebral hemorrhage, brain-tumor, cerebral abscess, tabes dorsalis, and multiple cerebrospinal sclerosis. **Central neuroses** also, especially hysteria, epilepsy, and neurasthenia, are frequently causes of vertigo. Vertigo is not rarely a result of **circulatory disorders** that directly affect the brain. Thus, the vertigo of the aged (senile vertigo) is probably dependent upon arteriosclerosis of the cerebral arteries. Vertigo is not unknown also as a symptom of conditions of anemia and exhaustion. At times vertigo has been observed to arise in the sequence of **traumatism** involving the skull, without the probability of anatomically demonstrable alterations in the brain. **Toxic vertigo** includes the cases in which the condition develops after excessive indulgence in alcohol, tobacco, coffee, tea, and opium. Vertigo may develop also in the sequence of **infectious diseases**, thus probably resulting from bacterial poisons—toxins. Under this heading belongs the vertigo of *malaria*, perhaps also so-called *Gerlier's disease*, which has been observed to occur in epidemic distribution in the vicinity of Geneva among shepherds and hostlers; a disease that, in addition to vertigo, is attended with ptosis, diplopia, amblyopia, and paresis of the flexors of the fingers and the legs. Vertigo may be induced intentionally by means of *galvanization of the skull* and adjacent parts—*electric vertigo*. Vertigo is often of **reflex** origin. Chronic disease of the stomach and the bowel causes vertigo with especial frequency, but the same condition occurs also in association with disease of the larynx, the heart, the sexual organs, the nose, and even with painful caries of the teeth. Vertigo is at times dependent upon **disease of the organs of special sense.** Paralysis of the ocular muscles is generally followed by vertigo. Foreign bodies in the ear, insufflation of air into the Eustachian tube, particularly, however, disease of the labyrinth, as well as that of the trunk of the auditory nerve, likewise causes vertigo. That variety of vertigo may be included in this group that appears upon staring at flowing streams, on jolting, on sea-voyages, in ascending high mountains, etc., and which probably is due to *deficient power of localization in space.* Occasionally no cause can be discovered for vertigo, and under such circumstances the condition has been spoken of as *essential vertigo.* In children vertigo occurs but seldom, as experience has shown.

Symptoms and Diagnosis.—Persons who are attacked by vertigo have a sensation either of being themselves moved in space or of their surroundings revolving about them. The movement may take place in a horizontal or in a vertical direction. During the attack of vertigo the patient may be seen to stagger, or in severe attacks to fall to the ground. Attacks of vertigo generally occur during the day, with especial frequency in the morning, when the stomach is empty; but some patients are seized with vertigo especially during the night in the course of a dream—nocturnal vertigo. The duration, the severity, and the number of attacks of vertigo are susceptible of infinite variation. The attacks of vertigo that occur in the sequence of disease of the labyrinth have been designated also *Ménière's disease.* In this disorder marked tinnitus aurium occurs. The patient becomes pale, and the skin is covered with a cold sweat. Often there are nausea and vomiting, occasionally nystagmus, and even slight loss of consciousness.

The **prognosis** and the **treatment** depend essentially upon the causative factors. Among symptomatic remedies bromids deserve the greatest confidence. In the treatment of Ménière's disease **quinin hydrochlorate** (0.5—7½ grains—thrice daily) has proved efficacious.

CATALEPSY.

Symptoms and Diagnosis.—Catalepsy is attended with attacks in which the patient is unable to execute voluntary movements, while passive movement of the members can be effected without resistance, and the attitudes thus induced may persist for several minutes or more, so that the movements are suggestive of those of a wax doll, and the condition has been designated *waxy flexibility of the muscles*. The *duration* of the attacks varies between minutes and days. The **eyes** are generally closed, the pupils dilated and unresponsive. **Consciousness** is lost in greater or lesser degree. The patients respond but sluggishly and feebly, if at all, to cutaneous or other irritation. The skin frequently feels cold, and is pale, while the temperature of the body may be elevated to 39° C. (102.2° F.). The conclusion of a cataleptic attack is often indicated by a deep sigh; consciousness is restored, and the patient generally has no knowledge of what has taken place. At times the disease is terminated with a single attack, but not rarely it persists for weeks, months, years, and even throughout the whole of life.

In contradistinction from **simulation**, it should be borne in mind that in malingerers tremor of the elevated member will take place within a short time, and this will soon be followed by a lowering of the member from muscular fatigue. **Involuntary muscular movements** (cardiac, respiratory, vesical, rectal, deglutitory) are executed without interference during the cataleptic attack. In cases of considerable duration there may be danger of death from inanition in consequence of involvement of the muscles of mastication in the rigidity, but if the food is introduced into the esophagus it will pass into the stomach without obstruction.

Etiology.—Catalepsy is an *uncommon disorder*, and it occurs with preference in *pallid* and *neurotic individuals*. It frequently appears in the course of **central neuroses**, particularly hysteria and epilepsy. It has been observed also in association with disease attended with anatomically demonstrable alterations, as, for instance, **meningitis, cerebral hemorrhage, cerebral softening,** and **tumor of the brain,** as well as with **progressive paralysis of the insane.** Occasionally it is a result of profound **emotional disturbances** (grief, fright, joy). *Toxic catalepsy* has been observed as a result of the inhalation of ether and of chloroform. Possibly *autotoxic catalepsy* occurs in cases of gout and nephritis. The designation *infectious catalepsy* has been applied to cases in which the disorder develops in the course of infectious diseases. It should further be mentioned that cataleptic states can be induced intentionally by **hypnosis** and **suggestion.**

Prognosis and Treatment.—The curability of the disorder depends upon the nature of the fundamental disease. During the cataleptic attack active **stimulating measures** should be employed (douches of cold water, inhalation of ammonia, tickling of the nose with a feather, etc.), and, with the termination of the attack, efforts should be directed to the relief of the primary disease.

CENTRAL NEUROSES IN WHICH SENSORY DISTURBANCES PREDOMINATE.

NERVOUS HEADACHE.

Symptoms, Diagnosis, and Prognosis.—The designation nervous headache should be applied only to such cases of headache as are *not dependent upon anatomically demonstrable disease of the brain and the cerebral meninges.* The disorder is generally a most troublesome one, and it not rarely persists from earliest childhood to advanced age, and is therefore known also as *habitual headache.* Often, naturally, it diminishes in severity in old age. The **character of the pain** is variously described, sometimes as boring, at other times as beating, occasionally rather as a sense of pressure in the head than headache. The **distribution of the pain** likewise varies. At times it is referred by the patient to the frontal region, at other times to the occiput, but not rarely it is distributed over the entire skull. At the time of the attack **nausea** and **vomiting** occasionally occur. Often the **scalp** is markedly tender to touch and when the hairs are pulled. The **face** is at times hyperemic and congested, and at other times pale and anemic, so that a distinction has been made between hyperemic and anemic nervous cephalalgia. Some patients are free from headache for scarcely a day, while in others it occurs only at long intervals, and particularly if mental over-exertion, excitement, gastric and intestinal disorders have preceded. In women it occurs frequently at the menstrual period. The *duration of the individual attack* varies between a few hours and an entire day; the pain often disappears after sleep.

Although headache is not a serious disorder, it incapacitates a good many persons from the pursuit of their work, and compels them to withdraw from society and seek seclusion in a quiet and if possible dark room until the attack has terminated. Some patients become morbidly reserved and hypochondriacal in consequence.

In the *differential diagnosis* the confusion of headache with an **anatomically demonstrable cerebral disorder** should be guarded against, and consideration should be attached especially to the presence of other general cerebral symptoms (inequality of the pupils, slowing of the pulse, papillitis). The pain due to **neuralgia of the nerves of the head** corresponds with the distribution of the affected nerves, in the course of which pressure-points can generally be discovered. **Inflammation of the bones of the skull** and their periosteum is generally attended with thickening, which is especially sensitive to pressure.

Etiology.—Headache is frequently an *inherited disease*, although it is not always transmitted as such, but alternates with other central neuroses, particularly hysteria, neurasthenia, and epilepsy. Often it is the result of **mental over-exertion** and **emotional disturbances.** It occurs frequently, therefore, in students, in men of learning, and in merchants. The headache of smokers and alcoholics, and that which follows excessive indulgence in coffee and tea, belong among the **toxic varieties of headache. Autotoxic headache** occurs in association with uremia, cholemia, gout, diabetes mellitus, gastric and intestinal disorders. **Infectious influences** are operative in the production of headache in the course of infectious diseases. Thus, severe headache occurs quite frequently at the commencement of typhoid fever and of syphilis. Occasionally headache results from **states of exhaustion.** It is, therefore, encountered in association with chlorosis, following loss of blood and suppuration, and after long vigils at night, and the like. **Headache of reflex origin** includes cases in which the condition occurs in conjunction with disease of the liver, the kidneys, the sexual organs, and other viscera. Occasionally headache is dependent upon **ocular disorders,** particularly errors of refraction and eye-strain.

Treatment.—Many patients have discovered definite means for the *relief of the attack of headache.* In some a darkened room, complete rest and seclusion, and a cup of strong coffee are serviceable; in others applications of cold water, or of water and vinegar; in still others marked compression of the head between the cooled hands, and the like. Among medicaments, **antipyrin, phenacetin,** and **sodium salicylate** are deservedly in good repute, and of any of these 1.0 (15 grains) may be administered thrice daily. One patient will be helped by one remedy and another patient by another remedy, and not rarely it becomes necessary to employ successively one remedy after another. Also, **antifebrin** (0.5—7½ grains—thrice daily) and **lactophenin** (0.5—7½ grains—thrice daily) have been recommended. Some patients obtain relief from the use of **migrainin** (a mixture of antipyrin, caffein, citric acid, in doses of 1.1—17 grains; if necessary, a second powder being given after an interval of two hours). The **migrain-pencil,** composed of *menthol,* also is praised by many patients. **Preparations of caffein** (caffein sodio-salicylate, caffein sodio-benzoate, 0.5—7½ grains—thrice daily, in powder) may also be mentioned. In order to prevent the recurrence of attacks of headache the causative factors must be carefully investigated, and **causal treatment** employed. Among **symptomatic remedies** the *bromids* have proved most serviceable in my experience, although it is necessary to employ them for a considerable length of time:

℞ Solution of sodium bromid,	15.0 : 200 (½ ounce: 6½ fluidounces);
Potassium bromid,	10 (2½ drams);
Ammonium bromid,	5.0 (75 grains).—M.

Dose: 15 c.c. (1 tablespoonful) thrice daily.

A sojourn in the country, among the mountains, or at sea may also be recommended.

HEMICRANIA (MIGRAINE).

Etiology.—Hemicrania, also designated migraine, is an exceedingly common and troublesome disorder, whose causative factors are essentially the same as those of nervous headache. *Heredity* is often an etiologic influence; but it is not necessary that all of the members of the family should suffer from hemicrania, for often the disorder alternates with hysteria, neurasthenia, epilepsy, psychopathy, and other central neuroses. Occasionally migraine occurs in the course of **central neuroses.** In addition to hysteria and neurasthenia, epilepsy should especially be mentioned. Often hemicrania precedes the development of epilepsy, or is a sequel of this condition, or it may replace the epileptic attack. **Mental over-exertion, anemia,** and **states of exhaustion** of all kinds, are not uncommon causes of hemicrania. **Toxic hemicrania** occurs especially in drunkards and smokers, while **autotoxic hemicrania** occurs in connection with gout. **Infectious hemicrania** has been observed also in the sequence of certain infectious diseases. **Reflex hemicrania** occurs in association with disease of the frontal sinuses, abnormal turgescence of the nasal mucous membrane, and diseases of the female generative organs, the liver, and the kidneys. Hemicrania often begins in childhood and persists throughout the whole of life. It rarely sets in after the twenty-fifth year of life. The disease is more common in *women* than in men.

Symptoms, Diagnosis, and Prognosis.—The principal symptom of hemicrania consists in pain distributed over one side of the head, which occurs paroxysmally, and, as experience has shown, more commonly upon the **left side.** Some patients awaken in the morning with this pain, while in others it sets in only in the course of the day. Occasionally an attack of migraine is preceded for days by **prodromes,** which consist especially in general malaise, disturbed sleep, loss of appetite, chilliness, and the like.

At times the attack of migraine sets in without demonstrable cause, while at other times it is preceded by emotional disturbances, alcoholic or venereal excesses, or gastric derangement. In women hemicrania occurs frequently with the onset of menstruation. In Zurich hemicrania occurs in many persons with the prevalence of the south wind.

The pain is described as boring, tearing, or beating, and its seat of greatest intensity is referred to the frontal, the temporal, or the parietal region, and less commonly to the occipital region. Often the pain extends down the nucha, and the patient experiences difficulty in movement and painful rigidity of the neck. Generally the scalp is exceedingly sensitive to slight touch, while deep pressure is much better borne. Traction upon the hairs generally induces great pain upon the painful side of the head. Frequently the patient complains of **nausea** at the time of the attack of pain, and **vomiting** occurs not rarely, the vomited matter being of watery consistency and containing an abundance of free hydrochloric acid, so that the existence of hypersecretion on the part of the gastric mucous membrane, and of hyperchlorhydria of the gastric juice, must be inferred. Some patients experience **rectal tenesmus**. After the attack a large amount of light urine is not rarely passed, which at times contains small amounts of albumin. Most patients are so overwhelmed by the pain that they are incapacitated for mental or physical exertion. In many slight mental confusion occurs, and in some instances I have even observed mild delirium. Generally, patients seek seclusion in a quiet, dark room and endeavor to sleep, because on awaking they generally find themselves free from pain. Often a sense of soreness and prostration persists for days after an attack. Many patients exhibit evidence of a threatening or an existing attack, as they appear pale and the face is drawn, the eyes hollow, and the skin is not rarely covered with a clammy sweat.

The *duration of an attack of migraine* is generally a few hours, but it often extends over an entire day, rarely longer. Some patients suffer from only a few attacks in the course of a year, while others are seized with a new attack at intervals of a few days. Cases are, however, on record also in which a new attack has set in before the preceding one had terminated, and under such conditions the designation *migrainous status* has been employed. Not rarely the *hair becomes course and gray* and *falls out* on the affected side of the head in the sequence of hemicrania. It is worthy of mention that at times the headache varies in situation in individual attacks—*alternating hemicrania;* or that it gradually passes permanently from one side of the head to the other. It may also happen that gradually the unilateral distribution disappears and the headache becomes diffuse.

In the *differential diagnosis* the same conditions must be taken into consideration as were mentioned on pp. 115 and 116 in connection with the diagnosis of nervous headache.

Vasomotor hemicrania, ophthalmic hemicrania, and the hemicranic equivalent should be mentioned as *special clinical varieties of hemicrania.*

In cases of **vasomotor hemicrania** the cervical sympathetic is obviously involved, for in the first place vascular changes are prominent upon the painful side of the head, and, in addition, the cervical sympathetic is tender on pressure. Two varieties of vasomotor hemicrania can be distinguished, accordingly as the vasomotor nerves are paralyzed or irritated, and the designations sympathetic-paralytic hemicrania and sympathetic-spastic hemicrania respectively are employed.

In cases of **sympathetic-paralytic hemicrania** the face upon the side on which the headache is present is greatly reddened; the corresponding carotid and temporal arteries exhibit marked fulness and pulsation. The skin of the face exhibits a higher temperature, and it is not rarely covered with sweat. The pupil is contracted—paralytic myosis. The palpebral fissure is narrowed and the eyeball is retracted; slight ptosis exists. The ocular conjunctiva is markedly injected, and hyperemia of the retina is visible in the fundus of the eye. Pressure upon the carotid artery not rarely mitigates the severity of the headache. The pulse is generally diminished in frequency. At the conclusion of the attack of pain the redness often is replaced by transitory pallor of the side of the face.

Sympathetic-spastic hemicrania is attended with pallor of one side of the face and smallness of the pulse in the corresponding carotid and temporal arteries. The affected half of the face is cooler. The pupil upon the same side appears dilated. Occasionally the secretion of saliva and of urine is increased, and the pulse is not rarely accelerated. Pressure upon the contracted carotid artery increases the unilateral headache, while pressure upon the carotid on the unaffected side affords relief. At the termination of the attack of pain increased fulness of the blood-vessels upon the affected side of the face generally takes place, and, in conformity therewith, increased redness of the face.

Ophthalmic hemicrania is characterized by the fact that the attacks of unilateral headache are preceded by *scintillating scotomata*, or these may appear only in the course of the attack. The appearances generally consist in bright zigzag figures suggestive of fortification-lines, whence the name *fortification-scotomata*. Often unilateral, but occasionally bilateral, hemianopsia and amblyopia also occur. At times other nervous symptoms are superadded, especially roaring and ringing in the ears, impairment of hearing, disorders of smell and taste, aphasia, agraphia, paraphasia, hemiparesis, hemiplegia, muscular spasm, hemianesthesia, hyperesthesia, paresthesia, excitability, depression, and mental confusion. Ophthalmic hemicrania especially exhibits intimate *relations to epilepsy*, which it may precede, or follow, or at times replace.

The **hemicranic equivalent** consists in attacks of vertigo and vomiting, spots before the eyes, aphasia, hemianopsia, mania, or mental confusion, which may occur instead of a well-developed attack of hemicrania or may alternate with it.

Treatment.—The treatment of hemicrania is governed by the principles that have been laid down on pp. 116 and 117 for the treatment of nervous headache.

CENTRAL NEUROSES IN WHICH VASOMOTOR AND TROPHIC DISTURBANCES PREDOMINATE.

VASOMOTOR NEUROSES OF THE EXTREMITIES.

Two varieties of vasomotor neuroses of the extremities are known, the *spastic* and the *paralytic*.

The **spastic vasomotor neurosis of the extremities** most commonly involves the fingers, the hands, and the arms, and occurs especially after prolonged *exposure to cold*. The condition is often encountered in washerwomen, so that it has been designated also *vasomotor neurosis of washerwomen*. The vascular spasm occurs paroxysmally, especially immediately after the action of cold. The patient experiences a sense of prickling, stinging, and formication—paresthesia—that frequently appears first in the tips of the fingers and then extends over the entire arm or only a portion thereof. The affected parts are pale and cyanotic, they feel cold, and the tactile sensibility of the overlying skin is lost in greater or lesser degree. Occasionally the skin is puffy. The muscles become stiff and weak, and the use of the arms is greatly impaired. The duration of an attack varies between seconds and minutes. The frequency of recurrence varies, as well as the duration of the entire disease. In some the disorder persists throughout life. Often but one arm is affected. If both upper extremities are involved, the symptoms frequently appear earlier and in more marked degree in one than in the other. At times an attack of vascular spasm is complicated by symptoms of stenocardia (vasomotor angina pectoris).

Spastic vasomotor neurosis of the extremities is a troublesome disorder, but unattended with danger, and it is best treated by abstinence from all work in cold water and in the cold generally, and by *spirituous frictions* of the skin, or by *brushing*, or application to the skin of the *electric brush* at the time of an attack.

In all probability related to spastic vasomotor neurosis of the extremities is the condition known as *acroparesthesia*, which is attended with prickling, burning, and painful sensations in the fingers and the toes in the absence of signs of vascular spasm.

Paralytic vasomotor neurosis of the extremities is known also as *erythromelalgia,* and occurs most frequently in the toes. The patient complains paroxysmally of severe burning and stabbing pains in the toes, particularly in the ball of the great toe, and the skin over the painful parts is greatly reddened and feels warm. Occasionally cerebral disturbances are superadded, such as vertigo, a sensation of fear, headache, pain at the nucha, impairment of vision, and mental confusion. The symptoms are aggravated by heat, and therefore during the *summer,* as well as by the upright posture. The disease occurs principally in *men* between the twenty-fifth and the fortieth year of life, who are *neurotic* and have suffered from *rheumatism, exposure to cold,* or *over-exertion.* Many physicians refer the *seat of the disease* to the lateral horns of the spinal cord. In the treatment of the disorder *immersion of the affected members in cold water, faradization, hydrotherapy,* and *residence in a uniformly mild climate* have been recommended.

INTERMITTENT VASOMOTOR ARTICULAR NEUROSIS (INTERMITTENT DROPSY OF THE JOINTS).

Intermittent vasomotor articular neurosis is attended with paroxysmal swelling of several joints, most frequently the knee-joints, in the absence of demonstrable inflammatory alterations and of pain in the affected parts. Such attacks may recur at fairly regular intervals of from four to six weeks, and may persist for as long as a week. In some patients the disorder continues indefinitely, while in other cases it disappears in the course of a few months. The affection occasionally occurs in the course of *exophthalmic goiter* and *vasomotor angina pectoris.* At times it has developed in the sequence of *malaria.* Occasionally only the *neurotic state* is elicitable as a causative factor. Of remedial agents, *iron, arsenic, quinin,* and *electricity* have been employed.

INTERMITTENT ANGIONEUROTIC EDEMA.

Intermittent angioneurotic edema is attended with circumscribed *edematous cutaneous swellings,* which are pale, less commonly reddened, generally the seat of prickling and burning, and most frequently situated on the extremities close to the large joints, although they may occur upon the eyelids, the cheeks, and the lips. In addition to the cutaneous edema *urticaria* occasionally occurs. *Articular enlargement* also sometimes takes place. Not rarely certain *mucous membranes* are involved in the edematous swelling. Changes in the pharyngeal mucous membrane are attended with difficulty in deglutition, and in the laryngeal mucous membrane with dyspnea, and this may occasionally attain dangerous intensity. The onset of the attacks is attended with *gastric derangement,* particularly vomiting of watery material, perhaps in consequence of edema of the gastric mucous membrane. *Pulmonary edema* also has been observed. *Constipation* is present at the time of the attack, and this often is followed by diarrhea with the termination of the attack. The *urine* occasionally contains albumin and blood. *Bleeding from the gums and the bronchi* also has been observed. The attack has often been preceded by exposure to cold. Some patients are never relieved of their disorder, while in other cases it disappears in the course of a few months. Danger may arise especially when the larynx is seriously involved. The disease is most common in *men.* The patients are often *neurotic individuals.* At times *hereditary transmission* has been observed. The disease has also occurred in the course of *exophthalmic goiter. Exposure to cold, alcoholic excess, rheumatism,* and *auto-intoxication* further are considered causative factors. The disorder has, for instance, been observed to develop after the ingestion of fish. In treatment *nervines* have been employed, and in the presence of marked swelling of the uvula *scarification* has been practised, and in the face of impending suffocation *intubation* or *tracheotomy.*

SYMMETRICAL GANGRENE.

Symmetrical gangrene is known also as *Raynaud's disease,* in honor of the physician who first described it. The disorder involves preferably the fingers and the toes, thus the peripheral portions of the body; but it occurs also upon the skin of the trunk and the face. The symmetrical distribution of the lesions upon the two sides of the body is noteworthy. The affected parts at once attract attention on account of their pallor, coldness, and numbness, in consequence of vascular spasm—so-called *stage of syncope.* This is followed by the *stage of asphyxia,* which is attributable to blood-stasis. The extremities acquire a bluish and reddish, mottled appearance, vesicles form upon the skin, which becomes black and gangrenous, and some phalanges may be exfoliated. Not rarely *fever, enlargement of the spleen,* and *albuminuria* are present. The disorder generally terminates in recovery. Occasionally repeated variations occur in its course. It may at times pursue an acute, at other times a chronic course. The disorder may be mistaken for ergot-poisoning (ergotism), *frost-bite, embolism,* and *thrombosis of the arteries of the extremities, senile gangrene, leprosy,* and *syringomyelia ;* and attention should be directed especially to the history, to arteriosclerotic changes, to the presence of valvular disease of the heart, to the demonstration of leprosy-bacilli and of partial anesthesia. Little is known with regard to the *causative factors.* Occasionally the disorder has made the impression of an independent *infectious disease*. At times it has occurred as a *sequel of other infectious diseases*. *Auto-intoxication* is probably operative in the development of the disorder in the course of diabetes mellitus. It has been observed also in cases of *hysteria, after emotional disturbances and neuritis*. *Women* are most commonly attacked by the disease. *Massage, electricity, nervines, iron*, and *preparations of cinchona* have been employed, and in addition *surgical intervention* may be required.

PERFORATING ULCER.

Perforating ulcer generally develops in the course of *nervous disorders*. At times it appears after *injury* or *inflammation of peripheral nerves*, at other times after *diseases of the spinal cord*, and at still other times after *diseases of the brain*. Most commonly the lesion is situated over the metatarsophalangeal joint of the great or the little toe, or over the heel. The epidermis at first becomes thickened, then becomes detached by serous or purulent fluid, and eventually drops off, while a generally round and sharply circumscribed ulcer remains, which appears as if cut out with a punch. The floor of the ulcer secretes but a small amount of thin secretion, and exhibits slight tendency to the formation of granulations and to cicatrization, so that occasionally the ulcer may persist for months. The destruction of tissue may extend to such a depth that joints are opened. In the vicinity of the ulcer cutaneous anesthesia often is present. Other trophic disturbances may readily occur in this situation, particularly abnormal growth of hair, desquamation of the epidermis, vesication, and the like. On microscopic examination of the skin endarteritic and neuritic alterations have been found. If the wound becomes the seat of septic infection, death may result from general septicemia. The *treatment* should therefore be directed essentially to the prevention of septic infection of the wound by the use of appropriate *bandages*. Occasionally the affected extremity has been amputated.

PROGRESSIVE FACIAL HEMIATROPHY.

Progressive unilateral atrophy of the face is a rare disorder, whose development is frequently preceded by *prodromes*, particularly pain and paresthesia on one side of the face, occasionally also vertigo and headache. The hair upon one side of the head also not rarely becomes light and gray, and falls out freely. The first appreciable changes consist in the formation of *oval, whitish*, or *brownish cutaneous areas* in the course of certain nerves, particularly the infraorbital nerve, upon one side of the face. Often areas originally whitish subsequently acquire a brownish hue. In these situations the skin is extremely thin, is wrinkled like a cicatrix, and the layer of subcutaneous fat particularly is found to be greatly wasted. *Cutaneous sensibility* is not rarely found to be impaired. These alterations result in great disfigurement, as the affected side of the face appears to be shrunken and the seat of cicatricial contraction (Fig. 19). The deformity becomes the more marked if the *bones of the face* also are involved in the atrophic process. Particularly the superior and the inferior maxilla and the malar bone frequently appear atrophied upon one side, and are conspicuous for their sharp bony processes. In the jaws the *absence of some of the teeth* has been observed. Excision and examination of the *muscles of the face* have disclosed that these also may be involved in the atrophic process. In any event, unilateral atrophy of the *tongue* and of the *muscles of the soft palate* not rarely occurs. The *cartilages of the nose and the ear* also are at times involved in the atrophic process. The *eye* upon the affected side is frequently retracted into the orbit, because the retrobulbar fatty connective tissue has disappeared, and the palpebral fissure appears at times contracted, at other times dilated. The *nasal* and *buccal orifices* also not rarely exhibit unilateral

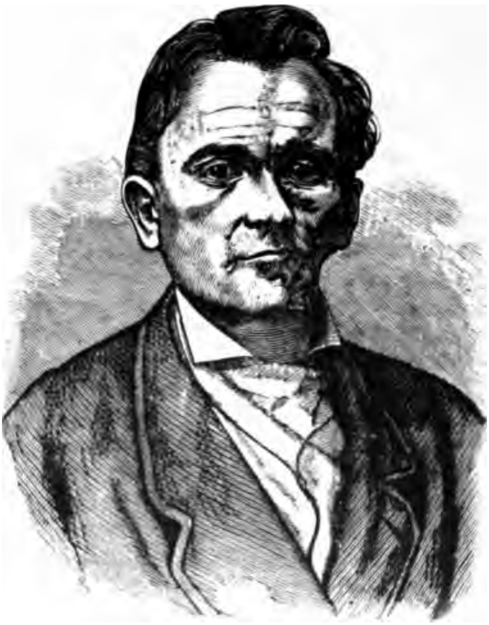


FIG. 19.—Appearance of the face in a case of left-sided progressive facial atrophy.

dilatation. Occasionally *symptoms of unilateral sympathetic irritation* (pallor of the skin of the face, dilatation of the pupils, and unilateral anidrosis) are observed. The disorder pursues a course of variable rapidity, and is followed by permanent disfigurement of the face. In one case the trigeminal nerve upon the affected side was found to be the seat of chronic proliferating neuritis, although the observation is so unique that it can scarcely be utilized in explanation of the disorder. Experience has shown that the affection occurs most commonly in *men*, and it generally sets in between the *tenth and the fifteenth year of life*. It begins but seldom after the twenty-fifth year. The patients are often members of *neurotic families*, and occasionally have previously suffered from *trigeminal neuralgia*, *spasm of the facial muscles*, *hemicrania*, or *epilepsy*. Unilateral atrophy of the face has been observed also in cases of *tabes dorsalis* and *multiple cerebrospinal sclerosis*. *Exposure to cold*, *traumatism*, and *infectious disease* also are mentioned as causative factors. The disorder is not dangerous, but incurable. Confusion with *congenital asymmetry of the face* is scarcely conceivable, as the condition under consideration is not a congenital one.

FACIAL HEMIHYPERTROPHY.

Unilateral hypertrophy of the face is a *rare disorder*, which occurs only as a *congenital* condition, and occurs with equal frequency in both sexes and upon both sides of the face. The soft parts of the face especially are involved in the hypertrophic process, but the tongue and the tonsil also have been hypertrophied on one side. Not rarely the arm and the leg also upon the same side of the body are involved in the hypertrophic process. At times the hypertrophied half of the face is the seat of abnormal redness, of dark discoloration of the skin, and of increased growth of dark hair. The sebaceous secretion often is increased, and it often collects upon the skin in small crusts. The disorder is incurable, and causes disfigurement of the face (Fig. 20), but it is unattended with danger to life.

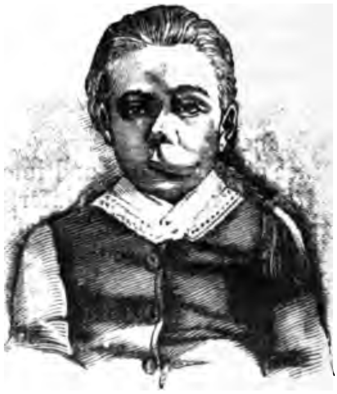


FIG. 20.—Left-sided facial hypertrophy in a girl 9 years old (after Schieck)

SCLERODERMA.

Symptoms, Diagnosis, and Prognosis.—Scleroderma is a rare disorder attended with **induration and contraction of the diseased portions of the skin.** Generally it appears first in the form of irregular **spots** and **bands,** which gradually increase in size and coalesce, and finally transform large areas of skin into firm and hard tissue. The changes occasionally develop insidiously, and often are discovered accidentally on palpating the skin or in consequence of alteration in the expression of the face or of interference with the mobility of the extremities; or the induration of the skin may be preceded by **swelling** and **redness,** and this has been designated the *edematous stage.* The diseased portions of skin exhibit thickening and condensation, so that their boundaries can be readily determined by passing the finger over them. The skin cannot be easily raised in folds, and is but slightly movable upon the subjacent tissues. The color of the skin is at times unaltered, at other times remarkably pale, and at still other times excessively pigmented. Occasionally an abundant deposition of pigment has taken place at the periphery of the spots. The sudoriferous and the sebaceous secretion often exhibits no change. Cutaneous sensibility also is preserved. The electric resistance of the skin has been found at times unaltered, at other times increased or diminished. The indurated portions of skin frequently become thinned and undergo cicatricial contraction, and various deformities and functional disturbances result. Extensive scleroderma of the skin of the face gives the **facial expression** a mask-like rigidity, causes impaired mobility and narrowing of the palpebral fissure, and often also the formation of fissures and contraction of the buccal orifice, so that the ingestion of food is seriously interfered with.

Linear and annular *scleroderma of the skin of the chest* causes a sense of constriction and interference with breathing. *Scleroderma of the extremities* often interferes with the mobility of the joints and gives rise to permanent deformities of the fingers, hands, and arms. Occasionally the fingers are involved earliest and in greatest degree, and often these alterations are preceded by the manifestations of a vasomotor neurosis and erythromelalgia. Under such conditions the individual phalanges become shortened, particularly the terminal phalanges, and some of the nails are exfoliated, and even the entire terminal phalanx. This condition has been designated *sclerodactyly* (Fig. 21). Often ulcers resistant to treatment appear upon the fingers. In some instances sclerotic changes have been observed also upon some of the **mucous membranes,** such as that of the mouth, the pharynx, the larynx, and the vagina.

Scleroderma generally pursues a chronic course, but not rarely exhibits numerous variations, the sclerodermatous areas becoming again soft and healthy. In general there is an undeniable tendency for the changes in the skin to extend progressively. The patients become exhausted and die as a result of marasmus. The *prognosis*, therefore, is not favorable, although there is no immediate danger to life. It may be mentioned, further, that occasional osseous and muscular atrophy and muscular sclerosis result in consequence of the compression exerted by the indurated and contracting skin.

Anatomic Alterations.—Microscopic examination of diseased portions of skin has disclosed increase and condensation of the connective tissue in the corium and in the subcutaneous connective tissue, thickening of the walls of the blood-vessels, and accumulation of round cells in the vicinity of the blood-vessels. Attention has recently been directed in several instances to atrophy of the thyroid gland, whence the theory has arisen that scleroderma is the result of an auto-intoxication originating from the thyroid gland, and that the disease is therefore genetically related to myxedema, exophthalmic goiter, akromegaly, and possibly also to unilateral atrophy of the face.

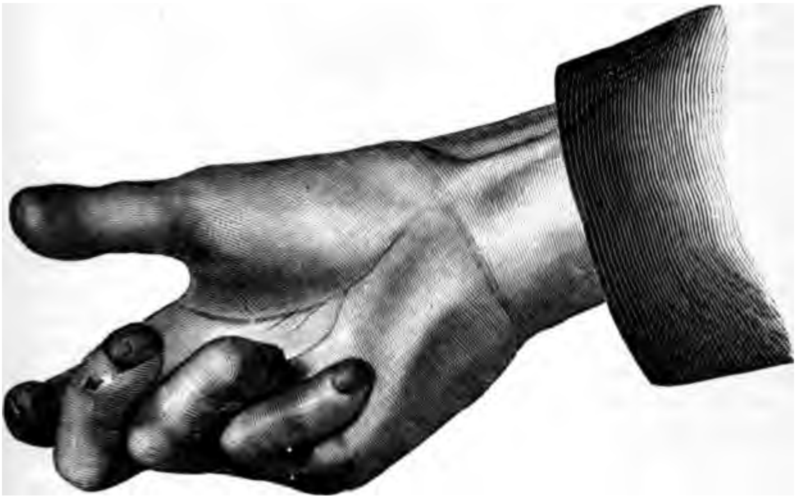


FIG. 21.—Sclerodactyly in a woman, 49 years old; from a photograph (personal observation, Zurich clinic).

Etiology.—Scleroderma generally develops *after the twenty-fifth year of life*, and occurs more frequently in *women* than in men. **Exposure to cold, traumatism, emotional disturbances,** and **antecedent erysipelas** have been mentioned among the causative **factors.** Occasionally the disease develops in the course of **exophthalmic goiter, unilateral atrophy of the face,** and **Addison's disease.**

Treatment.—Among **internal remedies,** successful results at times follow the administration of ***sodium salicylate*** **(1.0—15 grains—four times daily). Among external remedies,** ***massage***, *baths*, *galvanization*, and ***inunctions of oil*** **applied to the diseased skin may be recommended.**

MYXEDEMA.

Etiology.—Three varieties of myxedema must be distinguished, namely, the spontaneous, the operative, and the congenital. *Spontaneous myxedema* occurs most often in *women*, and generally develops after *sexual maturity*, but rarely in childhood or after the fiftieth year of life. The patients often are **neurotic individuals** or members of neurotic families. In isolated instances **heredity** was present; in others, a **familial distribution**—that is, the occurrence of the disease in several brothers and sisters. Exposure to cold, concussion, antecedent infectious disease, emotional disturbances, especially, however, profuse menstruation, pregnancy, parturition, and lactation are believed to be causes of the disorder. *Operative myxedema* has been observed after total, and occasionally also after partial, extirpation of the thyroid gland. The designation cachexia strumipriva or thyreopriva also has been employed. *Congenital myxedema*, finally, occurs in association with dwarfism and idiocy, and is known also as *sporadic cretinism.*

Symptoms and Diagnosis.—The principal symptoms of myxedema consist in **thickening of the skin, progressive cachexia,** and **nervous disturbances.** In cases of *spontaneous myxedema* the *thickening of the skin* is generally appreciated earliest in the **face,** which presents a peculiar disfigurement. The eyelids are converted into thick sacs and the eyes appear half closed. The **nose** becomes enlarged and the lips everted. The facial expression becomes stupid and dull (Fig. 22). At the same time the **skin** is pallid and alabaster-colored, and feels cool. The **sebaceous and sudoriferous secretions** are diminished, and often **desquamation of the skin** takes place. The **hair** falls out as well as the **teeth.** Gradually the thickening of the skin extends also to the **trunk** and the extremities. Of the latter the legs are generally involved earliest, and especially the toes are transformed into plump structures suggestive of the appearance of the toes of the pachydermata. It is noteworthy that, in contradistinction from ordinary edema, pressure with the finger leaves no depression in the skin. The gait of the patient is generally clumsy and waddling. Often the **tongue** becomes enlarged and speech slow and awkward. The **bodily temperature** is not rarely subnormal and the **pulse** slow. Some patients complain of **paresthesia.** Occasionally thickening of some of the **mucous membranes** (mouth, intestine, larynx) occurs, and when the larynx is involved the voice becomes markedly deepened. In diagnosis it is important to find the *thyroid gland* diminished in size, occasionally almost wholly absent. Gradually **anemia** and **exhaustion** become excessive. **Hemorrhage** readily takes place from various organs. Above all, **mental derangement** occurs (delirium, apathy, somnolence, and dementia). The disease pursues a *chronic course*, and occasionally does not terminate fatally for more than twenty-five years, amid progressive exhaustion.

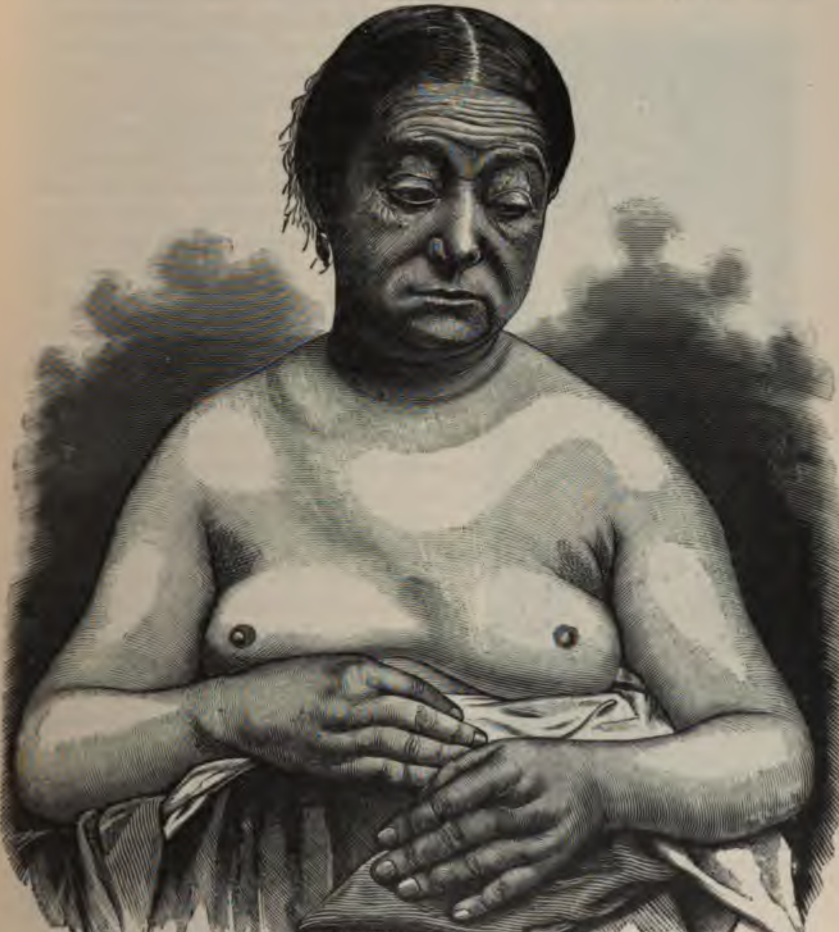


FIG. 22.—Facial expression in a case of myxedema (after Mosler).

Myxœdema strumiprivum generally develops some time after removal of the thyroid gland; occasionally it is preceded by tetany. The symptoms are identical with those that have been described. *Myxœdema congenitum* is attended, in addition to the symptoms described, with dwarfism, macroglossia, and idiocy.

Anatomic Alterations and Nature of the Disease.—Deposition of mucinous substances in the **skin** has been described. Occasionally cellular proliferation also has been demonstrable. Both in the skin and in the various internal viscera **obliterating endarteritis** has been encountered, in the liver and in the kidneys also interstitial connective-tissue hyperplasia. Tuberculous lesions have developed in the lungs with remarkable frequency. The alterations in the **thyroid gland** are of pre-eminent interest. In cases of both spontaneous and congenital myxedema the glandular tissue disappears, while the interstitial connective tissue increases progressively. It has been assumed, not without reason, that myxedema is the result of **auto-intoxication** dependent upon the deranged function of the thyroid gland. Possibly the thyroid gland should abstract from the blood and disintegrate certain substances, which, if retained in the blood in consequence of disease of the thyroid gland, cause intoxication and give rise to myxedema. Accordingly, certain relations would exist between scleroderma, exophthalmic goiter, akromegaly, and tetany, which also are referred to disturbances in the functional activity of the thyroid gland. In favor of the thyroid origin of myxedema also are the development of myxedema after operative removal of the thyroid gland, and the cure of myxedema by administration of preparations of the gland.

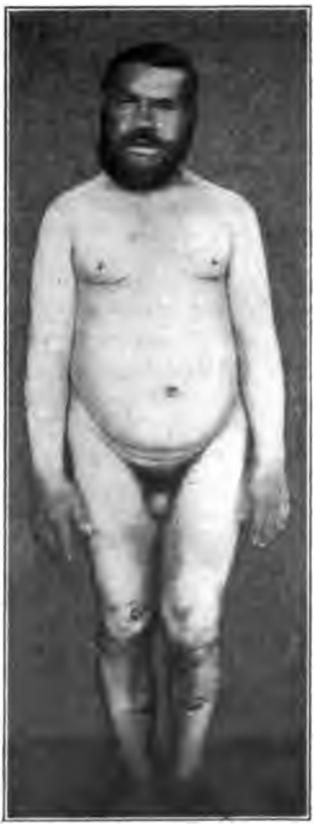


FIG. 23.—Myxedema (Cohen and Eshner).

Prognosis.—Although in recent times thyroid extract has proved an efficient remedy in the treatment of myxedema, the disease remains a serious disorder, as recurrence generally takes place as soon as the administration of thyroid gland is suspended.

Treatment.—**Prophylactic measures** are applicable in connection with operative attack upon the thyroid gland. Complete removal of the gland should, whenever possible, be avoided. Treatment of myxedema with **thyroid gland** constitutes a specific measure, and the only one to be recommended. *Thyroid tablets* constitute the most convenient means of administration, being manufactured on a large scale and easily secured. Further, certain precautionary measures should be observed. In the first place, the tablets should not be prepared from decomposed thyroid tissue, and, besides, they should not be administered in too large amount, as they may then readily give rise to palpitation of the heart, vertigo, albuminuria, glycosuria, and excessive emaciation, symptoms that have been grouped together under the designation of *thyroidism*. It is, therefore, necessary to intermit from time to time the administration of thyroid preparations. Some clinicians prefer tablets of *thyroiodin*, which Baumann has separated as the active ingredient of thyroid tissue.

EXOPHTHALMIC GOITER.

Symptoms.—The principal symptoms of exophthalmic goiter consist in **palpitation of the heart,**[1] **goiter, exophthalmos,** and **tremor.** *Palpitation of the heart—tachycardia*—is generally the symptom that first attracts the attention of the patient. At the beginning this often appears only after physical or mental exertion; but gradually it becomes persistent, and greatly annoys the patient in consequence of the distressing sense of pulsation in the chest, with which a sense of fear, constriction, and oppression becomes associated. The number of heart-beats is frequently increased from 60 to 80 to 120, and even much more, and the pulse occasionally can scarcely be counted. Often attacks occur, in the course of which the number of heart-beats is especially increased. At the same time the action of the heart is likely to be augmented. The chest-wall is, therefore, vigorously shaken by the cardiac impulse, a prominence gradually develops in the precordium, and the exaggerated activity of the heart is manifested by marked pulsation and throbbing of the carotids in the neck. Occasionally pulsation is visible in the palate, or palpable over the spleen or over the kidneys. Often dilatation and hypertrophy of the heart develop gradually. Systolic heart-murmurs also are not rarely audible. These are frequently of anemic or accidental origin, although occasionally a valvular lesion develops, most frequently insufficiency of the mitral valve. A cardiac-systolic arterial sound is frequently heard in the larger peripheral vessels. In the femoral artery a double sound has not rarely been observed.

In some instances the occurrence of palpitation of the heart has been preceded by *increase in the size of the thyroid gland,* while in other instances the latter follows or appears simultaneously with the cardiac palpitation. As a rule, both lobes of the thyroid gland share in the increase in size. The enlargement depends especially upon an increase in the glandular tissue, but the goiter is frequently rich in blood-vessels, so that on palpation a continuous thrill, increased with each cardiac systole, can be felt, and can also be heard on auscultation.

---

[1] It would seem preferable to employ the term "palpitation" to indicate the subjective perception of pulsation, and "tachycardia" to indicate accelerated action of the heart. Either may be present without the other.—A. A. E.

Exophthalmic goiter will especially attract the attention of the friends of the patient if **protrusion of the eyeballs or exophthalmos** (almost always bilateral) develops. The eyes project more and more from the orbits, and at times to such a degree that the lids are no longer capable of wholly covering the eyeballs. The latter condition is not without danger to the eye, as the cornea may readily undergo desiccation, foreign bodies, especially bacteria from the air, may find lodgment upon it and are not removed by closure of the lids and the secretion of tears, and thus ulceration of the cornea readily results, and may be followed by loss of the eyeball. When the exophthalmos is marked the eyeball can be moved with difficulty, and insufficiency of the ocular muscles

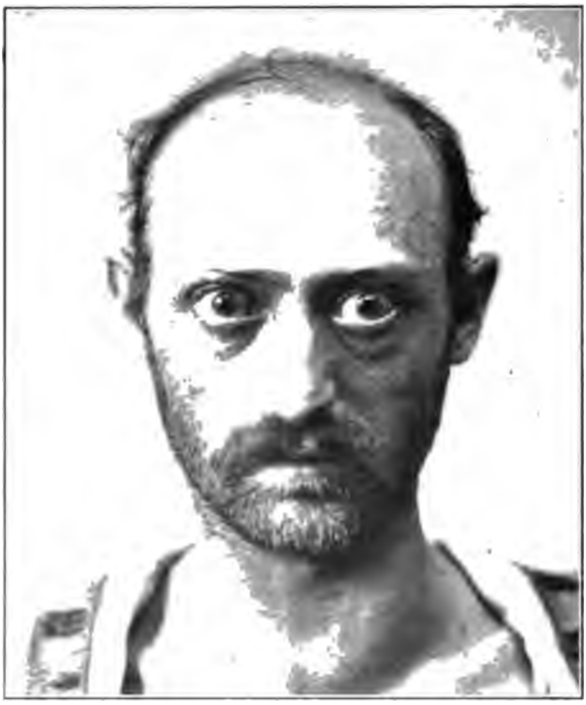


FIG. 24.—Case of exophthalmic goiter in a male (Cohen and Eshner).

develops. Convergence of the eyes, especially, cannot be maintained for any length of time. Advanced protrusion of the eyeballs causes marked disfigurement of the face. The patient acquires a surprised, frightened, and perplexed expression.

Occasionally the *Graefe symptom* is encountered, which consists in incongruity between the movements of the eyeball and the upper eyelid. If the patient be requested to fix his vision upon a finger held above his head, and to follow carefully the movements of the finger with the eyes, and the finger is suddenly displaced downward, the eye will follow more quickly than the upper lid, so that the latter remains behind the former. *Stellwag's symptom* consists in the faculty on the part of patients with exophthalmic goiter to keep the eyes open for a long time, without a sense of the need of frequent winking. The *pupils* occasionally are unequal, generally react well to light, and are at times unchanged in size, although at other times dilatation or contraction has been reported. Examination of the fundus has often disclosed *pulsation of the retinal arteries.*

The patients generally suffer from *tremor*, which is usually fine and rapid (from eight to nine and a half movements in the second).

Among the typical symptoms of exophthalmic goiter *diminution in the electric resistance of the skin* has recently been described, but this phenomenon is not directly related to the disease, but to the fact that sweating is generally profuse, and a moist skin is a better conductor for the electric current.

Individuals with exophthalmic goiter are often *pallid, blond, blue-eyed,* and *delicate persons*, in whose temperament a *neurotic element* is generally discoverable. They are excitable, and not rarely complain of **sleeplessness.** Other nervous disturbances also are often present. Generally slight **sweating** occurs. **Increased secretion of saliva and of urine** also has been observed. Occasionally **scleroderma, circumscribed alopecia, circumscribed cutaneous edema, urticaria,** or **articular swelling** appears. Often attacks of profuse watery **vomiting** and alarming, profuse **diarrhea, neuralgia, paralysis,** and **muscular atrophy** also have been reported. Occasionally **nuclear paralysis** of a number of cerebral nerves occurs. Now and then myxedema or akromegaly has been superadded to exophthalmic goiter. Occasionally psychopathies have developed. At times exophthalmic goiter is complicated by **glycosuria,** or even by **diabetes mellitus.** In the majority of cases exophthalmic goiter pursues a *chronic course*. Often periods of remission and exacerbation alternate with each other. Aggravations occur with especial readiness after physical and mental exertion. The disease is generally unattended with fever. The principal danger consists in progressive exhaustion and in insufficiency of the heart-muscle, with progressive general venous stasis.

Diagnosis.—The recognition of exophthalmic goiter is easy if the typical symptoms are kept in mind. Nevertheless, similar conditions may occur in anemic individuals. In these, goiter may develop, and this, by pressure upon the veins of the neck, causes protrusion of the eyeballs, and by pressure upon the cervical sympathetic induces also palpitation of the heart. Tremor and other nervous disturbances, however, are generally wanting.

Etiology and Nature of the Disease.—Exophthalmic goiter occurs *most frequently in women*, and childhood is generally exempt. Members of *neurotic families* or *individuals with acquired neurotic tendencies* are most readily attacked. Occasionally exophthalmic goiter develops in the course of certain **nervous diseases**, as, for instance, hysteria, tabes dorsalis, or multiple cerebrospinal sclerosis. Some observations in which **heredity** was a factor are also on record. Without doubt **emotional disturbances** are of etiologic significance. I have observed exophthalmic goiter to develop also after **excessive physical exertion**, as, for instance, after mountain-climbing. At times **infectious diseases** have been antecedent conditions. The disease is thought to arise through **reflex influences** in the presence of morbid distensibility of the erectile tissues of the nose, of disease of the female generative organs, and after pregnancy and parturition. In many instances no causative factor can be elicited.

As *anatomic alterations* peculiar to exophthalmic goiter are not known, free play has been given to hypotheses as to the *nature of the disease.* According to recent conceptions, the disorder is the result of *auto-intoxication* dependent upon morbid increase in the functional activity of the *thyroid gland—hyperthyroidism*—although nothing definite is known with regard to the nature of the disturbance. It is also not known what parts of the central nervous system are especially affected by the toxic process. Formerly the symptoms were attributed to disease of the sympathetic, but at present it is suggested that the medulla oblongata may be affected. In my opinion, various portions of the central nervous system may be involved. If this view be correct, the disease will have certain relations to scleroderma, myxedema, akromegaly, and tetany.

Prognosis.—The prognosis of exophthalmic goiter is not favorable, as permanent recovery is uncommon, and death may result from excessive weakness of the heart-muscle.

Treatment.—**Causal treatment** is scarcely ever applicable. Cauterization of the turbinate bodies of the nose is said to be capable of effecting a cure if the mucous membrane is unduly swollen. In the **symptomatic treatment** the *recumbent posture*, *milk-diet*, and *constant application of an ice-bag to the precordium* may be recommended. Among drugs, preparations of iron and iodids are especially to be advised; for instance:

R Sirup of iron iodid,
  Simple sirup, each, 50.0 (1⅔ fluidounces).—M.
Dose: 5 c.c. (75 minims) thrice daily.

℞ Solution of sodium bromid, 15.0 : 200 (½ ounce : 6½ fluidounces);
Potassium bromid, 10.0 (2½ drams);
Ammonium bromid, 3.0 (45 grains).—M.
Dose: 15 c.c. (1 tablespoonful) thrice daily.

All possible *nervines*, *courses of treatment with cold water*, *electricity*, and *massage* have also been employed. Recently *removal of the thyroid gland—thyroidectomy*—and *ligation of the thyroid arteries* have been practised. It cannot be denied that after such operations the palpitation of the heart and the protrusion of the eyeballs became less, and even disappeared; but other symptoms, such as tremor and a tendency to sweating, generally persist. Some clinicians have observed material improvement after *bilateral resection of the cervical sympathetic*. Treatment with *thyroid tablets* usually aggravates the symptoms. Thymus-tablets are said to be occasionally useful.

Digitalis and its substitutes are generally not successful in controlling *palpitation of the heart*. For the relief of the *enlargement of the thyroid gland* applications of tincture of iodin or inunctions of potassium-iodid or iodoform ointment may be employed. Inunction of the eyelids with potassium-iodid ointment has also been recommended for the *protrusion of the eyeballs*. Excessive exophthalmos may require the application of protective bandages to the eye or tarsorrhaphy.

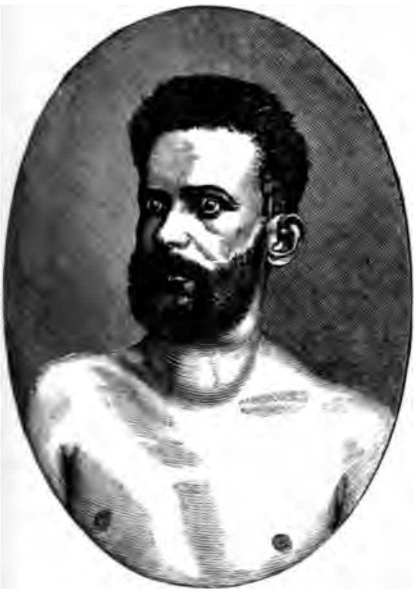


FIG. 25.—Facial expression in a case of exophthalmic goiter in a man 23 years old; from a photograph (personal observation, Zurich clinic).

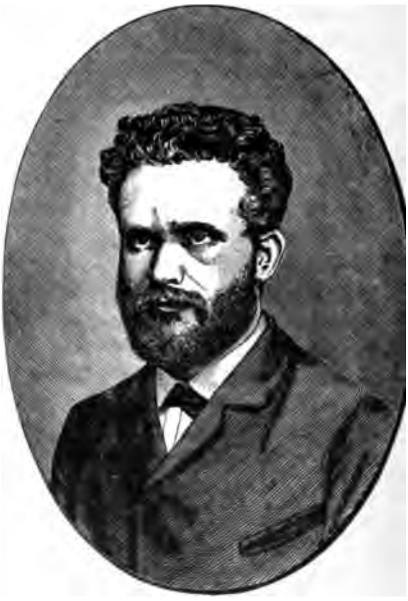


FIG. 26.—The same patient two months before the onset of an attack of exophthalmic goiter of ten months' standing.

AKROMEGALY (MORBID GIANT-GROWTH).

Symptoms and Diagnosis.—Akromegaly is attended with abnormal increase in size of the extremities of the bones, particularly of the hands, the feet, and the lower jaw. The disorder occasionally begins with **prodromes**, consisting in vertigo, headache, and paresthesiæ, and sometimes preceding the alterations in growth for a number of years. As a rule, the disorder does not begin until after the completion of puberty. The abnormal enlargement commences at times in the hands, at other times in the feet, and at still other times in both sets of members simultaneously, and involves at times one finger or toe, at other times several fingers or toes simultaneously. The affected parts increase in length, but particularly in thickness, and acquire a sausage-like or paw-like appearance. The attention of the patient is often attracted to the changes by the fact that he must obtain gloves and also shoes of progressively increasing size. The bones of the hands and the feet also, as well as those of the forearms and the legs, less commonly those of the upper arms and the thighs, take part in the abnormal increase in length and thickness. Upon the leg there are occasionally associated also thickening and brownish discoloration of the skin, so that the member resembles that of an elephant. The thickening generally is especially marked at the lower epiphyseal extremities.

The patient complains not rarely of **paresthesiæ**, although cutaneous sensibility is unaltered. The **knee-jerk** has occasionally been wanting. The **muscles** generally remain unaffected, and atrophy occurs but seldom. Changes in the **electric irritability** do not take place. In the **face**, abnormal enlargement occurs especially in the **lower jaw**, so that this member projects markedly forward and the margin of the contained teeth is situated in advance of that of the teeth in the upper jaw. To [illegible] the inferior maxilla appears [illegible] and thickened. The **nose** also becomes enlarged, and [illegible] of eversion of [illegible]. The **eyelids** are enlarged [illegible] cartilages of the ear, so that the- [illegible] dis- [illegible] the [illegible] **kyphosis** [illegible] **soft parts** [illegible] in [illegible] **lips** [illegible] **tongue** [illegible] in size, plump, and awkward, and speech appears slow and difficult. The **larynx** becomes unusually large, and the **voice** acquires a strikingly deep and rough character. The **organs of generation** also undergo change; the penis and the clitoris attain extraordinary length, and the vagina becomes excessively dilated and capacious. The **heart** and the **arteries** also attain considerable size, as has been demonstrated especially on post-mortem examination. It is a distinctive feature that **changes in the sexual functions** occur quite constantly: in men, diminution in sexual vigor and impotence; in women, cessation of menstruation. The thyroid gland is frequently, but by no means constantly, atrophied; while frequently dulness on percussion has been present

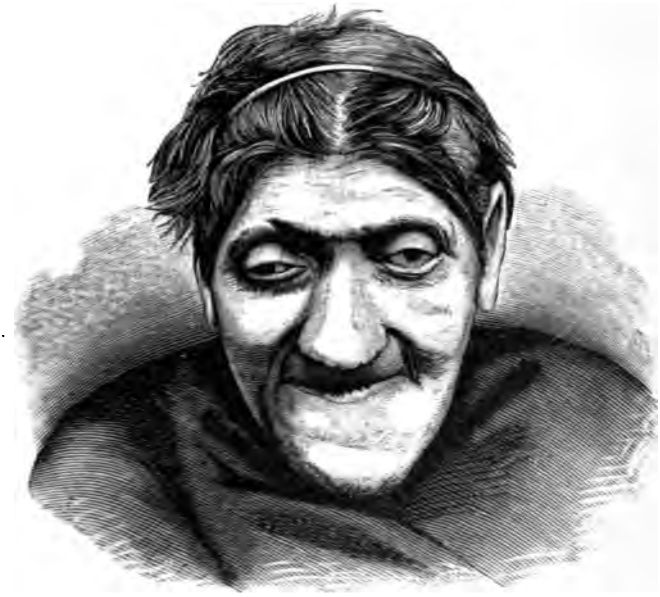


Fig. 27.—Facial expression in a case of akromegaly (after Marie).

over the manubrium sterni in consequence of *persistence of the thymus gland.* **Disorders of vision** also occur often, in consequence of enlargement of the pituitary body and the resultant pressure upon the optic nerve. Unilateral or bilateral contraction of the visual field, hemianopsia, amblyopia, and amaurosis have been observed, and in association with hemianopsia and hemianopsic reflex rigidity of the pupil. Ophthalmoscopic examination not rarely discloses inflammation or atrophy of the optic nerve. **Cerebral disturbances** are frequently encountered, particularly headache, vertigo, progressive loss of memory, and dementia. The disease may continue for many years, and often terminates amid symptoms of excessive exhaustion. Polyphagia, polydipsia, polyuria, diabetes mellitus, alimentary glycosuria, albuminuria, albumosuria, and chronic nephritis have been observed as *complications.*

Diagnosis.—Morbid giant-growth is distinguished from normal giant-growth by the fact that the latter begins in youth, involves all parts of the body in like manner, is not confined principally to the hands, the feet, and the lower jaw, and is not unattended with nervous symptoms. **Elephantiasis** and **myxedema** are attended with thickening of the soft parts, particularly the skin, while the bones remain unchanged. **Leontiasis ossea** is attended with the formation of nodular excrescences upon the cranial bones, while the extremities remain exempt. **Deforming osteitis** is attended with enlargement of the extremities, but the latter are painful and become deformed, while the hands and the feet particularly escape. **Deforming polyarthritis** is attended especially with changes in the joints, while these parts remain uninvolved in cases of akromegaly. **Hypertrophic osteo-arthropathy** is attended with bulbous, drumstick-like thickening of the terminal phalanges of the hands and the feet, which develops especially in connection with chronic disease of the air-passages and congenital lesions of the heart, while nervous symptoms, alterations in vision and in sexual power, kyphosis, and changes in the lower jaw are wanting. Occasionally **partial giant-growth** is observed, involving only a few fingers or toes. If the condition is congenital, it is designated macrodactyly or macropodia respectively. Acquired partial giant-growth occurs in association with syringomyelia and neuritis.

Etiology.—Akromegaly is an *uncommon disease*, which experience has shown occurs *more frequently in men* than in women. Isolated observations of its **hereditary** and **familial occurrence** are on record. Not rarely the disorder has occurred in members of neurotic families, themselves suffering from central neuroses, as, for instance, **hysteria, myxedema,** or **exophthalmic goiter.** Akromegaly has been observed also in the sequence of **tabes dorsalis.** It has followed **infectious diseases** (syphilis, articular rheumatism, small-pox, malaria). The disease has been attributed also to **gout, alcoholism,** and even **fright.**

Anatomic Alterations.—The affected **bones** are thickened, although their vascular canals are dilated. Occasionally the bones are the seat of hyperostoses, and at times the articular extremities also are changed. The internal **viscera** are occasionally all increased in size, and exhibit hyperplasia of the interstitial connective tissue and hyaline degeneration of the smaller blood-vessels. The **thyroid gland** is often, but not constantly, atrophied or goitrous. **Hypertrophy of the pituitary body** is almost always present, and at times has caused a deep excavation in the sella turcica, and often has exerted pressure upon the adjacent optic nerves. **Per-**

sistence of the thymus gland has been observed in numerous instances.

Nothing is as yet known with certainty as to the *nature of the disease.* By some it is considered the result of *auto-intoxication,* originating in the thyroid gland. The hypertrophy of the pituitary body is accordingly to be looked upon as compensatory (vicarious) for the loss of the thyroid gland. The by no means constant persistence of the thymus gland is opposed to the thymic origin of the disease. As nothing is known concerning the function of the pituitary body, it is venturesome to attribute auto-intoxication to this gland.

Prognosis.—The disorder is resistant to all treatment, but it is unattended with immediate danger to life.

Treatment.—**Organotherapy** has, as yet, scarcely yielded successful results. In a case under my observation tablets of thyroid gland were equally inefficacious with tablets of pituitary body and thymus gland. Symptomatic treatment must therefore be resorted to.

CENTRAL NEUROSES IN WHICH PSYCHIC ALTERATIONS PREDOMINATE.

HYSTERIA.

Etiology.—Hysteria is an exceedingly *widespread disease,* which for a long time was erroneously thought to occur almost exclusively in *women.* As a rule, it does not develop before puberty, although hysterical prodromes may frequently be traced into *childhood,* and every now and then it happens that even children are the subjects of well-developed hysteria. The more active and the more irritable the nervous system, the more readily may hysteria develop. The disorder is therefore encountered with especial frequency among certain *nationalities,* as, for instance, the French and the Jews. Hysteria occurs with extraordinary frequency as an **hereditary disorder,** so that a number of clinicians of wide experience have adopted the view that heredity alone is of etiologic significance. Hysteria frequently develops in the course of other **nervous diseases,** among which chorea and multiple cerebrospinal sclerosis may be mentioned. Hysteria is often the result of improper **education** and **mode of life.** Children subjected to excessive mental strain are attacked by hysteria during childhood, or perhaps later. Excessive mental activity, morbid ambition, and the like, also give rise to hysteria in adults. At times hysteria is due to profound **emotional disturbance.** Anxiety, grief, unfulfilled hope, home-sickness, and disappointment in love may be mentioned in this connection.

All **debilitating** and **protracted diseases** may be followed by hysteria. I have often observed hysteria develop during convalescence from severe typhoid fever, but a similar result may take place in the sequence of chronic disease of the stomach, the intestines, the liver, and the kidneys, and of some other viscera. It was formerly believed that diseases of the uterus particularly, and especially displacements, constituted a frequent cause for hysteria, and the name of the disease was selected in accordance with this view. The **toxic variety of hysteria** includes the cases that develop after excessive indulgence in alcohol, tobacco, coffee, and tea, and chronic lead-poisoning. At times **imitation** plays an important [illegible] in the etiology. Individuals who live or associate with hysterical patients are not rarely themselves attacked by hysteria. It therefore happens that hysteria may occur in endemic distribution in closed institutions (schools, boarding-houses, convents). Often the hysterical tendency has lain dormant in an individual for a long time before it has been wakened into activity by a definite [illegible]. Particularly profound emotional disturbances [illegible] hysterical symptoms.

Symptoms. [illegible] hysterical symptoms [illegible] important part, although by [illegible] actual ex- [illegible] suffers with [illegible] himself [illegible] will it [illegible] disturbances in [illegible] all [illegible] motor, [illegible] symp- [illegible] the laryngeal muscles are frequently paralyzed, particularly the posterior crico-arytenoid and the internal thyro-arytenoid.

Among the less common manifestations of hysteria is *paralysis of the pharynx and the esophagus*, rendering deglutition difficult or impossible, and if long continued requiring feeding by means of the stomach-tube. There may be also *paralysis of the detrusor of the bladder*, with consequent retention of urine, or *paralysis of the sphincter of the bladder*, with consequent incontinence of urine.

Clonic and *tonic muscular spasm* occurs quite frequently in hysterical patients, and is susceptible of all possible variations with regard to distribution, duration, and severity. Often blepharospasm, torticollis, and spasmodic tic are dependent upon hysteria. Not rarely patients complain of a sense of constriction ascending from the epigastrium or the hypogastrium. This is known as *hysterical globus*, is probably dependent upon ascending spasm of the muscular layer of the esophagus, and is an important diagnostic symptom. Reference may be made in this place, further, to hysterical *tremor*, *choreiform motor disorders*, *ecstatic manifestations*, *catalepsy*, and *myoclonus*.

Muscular contractures in the arms involve preferably the flexors; in the legs the extensors. Occasionally contracture of the masseters gives rise to *hysterical trismus*, unilateral contracture of the muscles of the nucha to *wry-neck*; but *contracture of the muscles of the tongue* upon one side, with deviation of the tongue to one side, and *curvature of the vertebral column* as a result of contracture of the muscles of the back, also are known.

Sensory disorders constitute a frequent manifestation of hysteria, especially **cutaneous anesthesia.** The latter is at times confined to a few circumscribed areas, which scarcely coincide with the anatomic distribution of the cutaneous nerves; while at other times it involves certain portions of the extremities in an annular or band-like manner, or it may involve entire extremities or portions of extremities, or occur in unilateral distribution—hysteric hemianesthesia—or extend over the entire body. Not rarely circumscribed anesthesia moves from place to place within the shortest possible time. It is a remarkable fact that occasionally it is possible by means of certain devices to displace anesthesia from one side of the body to a corresponding point upon the other side; and the phenomenon is designated *transference*. This may be effected by the application of mustard-plasters, bone-plates, or coins upon the anesthetic or upon the unaffected skin. Occasionally the skin is the seat of **hyperesthesia.** This may coexist or alternate with anesthesia. Not rarely circumscribed areas are especially sensitive to pressure, with particular frequency the region of the ovaries—so-called ovaria. Pressure upon such points may induce hysterical attacks, but it may also suppress developed attacks. **Neuralgia** is often of hysterical origin. Many patients complain also of **paresthesiæ** of most diverse character. Often pain at the summit of the head is referred to—hysterical clavus—or of a feeling of a cold body—ovum hystericum. Occasionally pain appears in the joints—hysterical articular neurosis. Frequently some of the **mucous membranes** are involved in the sensory disturbances. Anesthesia of the pharyngeal mucous membrane is encountered with especial frequency in cases of hysteria. Paresthesia of the laryngeal mucous membrane is frequently encountered, giving rise to hysterical cough and difficulty in articulation.

The **vasomotor** and **secretory** disorders include pallor and abnormal redness of the skin, increased secretion of saliva, and changes in the secretion of urine. Occasionally the secretion of urine is suppressed almost wholly for days—hysterical anuria; and it may be replaced by profuse watery vomiting, in which large amounts of urea have been demonstrated; or the patients may pass unusually large amounts of urine within a short time, the secretion being generally watery, limpid, and of low specific gravity—*spastic urine*. Secretory disorders involving the gastric and intestinal mucous membrane may also occur, giving rise to *hypersecretion* and *hyperchlorhydria of the gastric juice, mucous diarrhea*, and *membranous enteritis*.

Occasionally cutaneous blood-vessels rupture and an extravasation of free blood takes place upon the skin; and this condition has improperly been designated *blood-sweating—hemidrosis*. This phenomenon is of significance because religious importance has been attached to it, and it has been considered as a miraculous manifestation. At times *osmidrosis* has been described in hysterical patients, the sweat, for instance, giving off the odor of violets or of musk. Occasionally hemorrhage takes place from the gastric or the bronchial mucous membrane, with the occurrence of hysterical hematemesis or hemoptysis.

Trophic disorders occur but rarely in hysterical patients, and must be viewed with great suspicion. All hysterical patients have a morbid tendency to solicit the interest of their friends and of the physician; and in order to rouse and to maintain this they not rarely resort to artifices and deceptions. They are especially fond of **self-mutilation**, which they secretly accomplish with great ingenuity, so that an inexperienced and careless clinician might be readily deceived. Of the **organs of special sense** the *eye* suffers with especial frequency. *Contraction of the visual field* both for white and for colored light, especially for green, is particularly common. *Amblyopia* and *amaurosis* also occur, the latter upon one side and often without the consciousness of the patient. Occasionally unilateral *impairment of hearing* or *deafness* develops, and ringing or roaring in the ears, and the like, readily occur. Hyperosmia, anosmia, parosmia, hypergeusia, parageusia, and anageusia are likewise not unknown as symptoms of hysteria. Occasionally disorders of certain viscera occupy a conspicuous position; thus, there may be asthmatic states, attacks of palpitation of the heart, singultus or eructation, gastralgia, enteralgia, borborygmi, tympanites, and the like.

Hysterical patients generally exhibit **changes in their psychic functions.** At times they are abnormally irritable, while at other times they are indifferent and apathetic. At times well-developed states of mental derangement occur. The patient exhibits capriciousness, is morbidly introspective, and displays a tendency to exaggeration, falsehood, and deception. The mental equilibrium is readily disturbed on slight provocation, with the development of attacks of laughter or weeping or screaming. The spirit of unwillingness—*aboulia*—is of great significance in the development of hysterical manifestations. Some patients maintain that they are unable either to stand or to walk—*hysterical abasia* and *astasia*—although they are capable in the recumbent posture of executing vigorous muscular movements. Others refrain from walking for years because attempts at walking are attended with severe pain—*akinesis algera.* The aboulia is responsible also for *hysterical mutism. Hysterical aphasia* is not to be confounded with mutism. The hysterical disorders of speech include also *hysterical stuttering* and *hysterical anarthria. Hysterical convulsions* are worthy of especial mention; these have been designated also *hystero-epilepsy.*

The severity, the duration, and the recurrence of the attacks exhibit extremely wide variations. The clinical picture is in many respects suggestive of epilepsy, particularly on account of the clonic muscular spasm, which involves the entire body with great violence. In contradistinction from epilepsy, however, consciousness is generally preserved, or is only obscured, without being completely lost; likewise, the pupillary reactions are unchanged; the tongue is bitten but rarely; and, finally, the patients throw themselves about more violently, cry and groan generally throughout the entire attack, tear their clothing and the bed-linen, and do not fall to the ground involuntarily at the beginning of the attack, so that external injuries are but rarely inflicted. The French clinicians especially have described as major hysterical attacks convulsions of which definite stages can be distinguished, clonic muscular spasm generally following a stage of hysterical distortion (clownism), which passes over into a stage of passionate attitudes, and finally is followed by hallucinations. The hysterical convulsion is occasionally followed by a post-paroxysmal state, in which the patients have no knowledge of their acts, and commit crimes of which they have no intimation when consciousness is restored.

Hysteria occasionally persists throughout the whole of life, but under such circumstances periods of improvement and aggravation alternate with each other, the latter generally being induced by emotional disturbances. Some patients, however, are freed from their disorder if relieved of obstinate diseases, if long-cherished hopes are fulfilled, if placed amid more favorable educational surroundings, and like conditions.

Diagnosis.—It is by no means always easy to recognize hysterical symptoms with certainty. In favor of an hysterical origin are the remarkably rapid variation and the sudden disappearance of nervous symptoms. It is important to look for *hysterical stigmata*, including especially anesthesia of the pharyngeal mucous membrane and abolition of the pharyngeal reflex, contraction of the visual field, circumscribed anesthesia, hysterical globus, and hysterical painful points. The clinician with large experience will often suspect hysteria from the general demeanor of the patient.

Prognosis.—Hysteria is of itself unattended with danger to life, but the patients are a source of anxiety, less to themselves than to those by whom they are surrounded and whom they harass and occasionally tyrannize over. Not rarely hysterical patients attempt suicide, but the effort is not serious; at any rate, they are always able so to arrange matters that the attempt is known of in advance and is prevented. Self-mutilation also is usually of such a character that life is not endangered thereby.

Treatment.—In the treatment of hysteria, **causal therapy** occupies the first place. Under all circumstances, appropriate *mental treatment* will yield the best results; and the more readily if the patient is removed from unsuitable surroundings. The physician should endeavor to secure the entire confidence of the patient, and to utilize his influence in every possible favorable way. Under some circumstances a certain degree of brusqueness will be unavoidable. Especially in cases of hysteria, the success of treatment will depend essentially upon a proper attitude and tact on the part of the physician. With medicaments, among which nervines particularly are to be considered, with electricity, courses of treatment with baths, and massage, little will be accomplished. It is above all of importance to fortify the will-power of the patient, and to overcome his unwillingness. If **hypnosis** and **suggestion** are successful, they often yield brilliant results.

NEURASTHENIA.

Etiology.—Neurasthenia depends upon abnormal irritability and undue readiness of fatigue on the part of the nervous system. **Heredity** is of great importance in the development of neurasthenia, and in this connection not alone this disease, but also other central neuroses, or anatomically demonstrable disease of the nervous system, may occur in the families in question. Occasionally the neurasthenic predisposition is **congenital**. The children of alcoholic parents suffer from neurasthenia with especial frequency, as well as those of parents who have married late in life or who at the time of conception have been debilitated by severe disease (syphilis, pulmonary tuberculosis, carcinoma). In many cases, however, neurasthenia is an *acquired disease.* Often the basis for the disease is laid in youth, particularly by mental overwork at school, excessive stimulation of ambition, neglect of bodily care, onanism, and the like. In addition to onanism, sexual excesses of other kind constitute a frequent cause for neurasthenia. Neurasthenia is often the result of **excessive mental activity.** It is therefore encountered with especial frequency in those engaged in intellectual pursuits, as, for instance, scholars, merchants, actors. Frequently candidates for examination become neurasthenic, in consequence of mental over-exertion and, in addition, of a fear of the results of the examination. Under certain conditions the earliest neurasthenic symptoms develop in the sequence of **infectious disease,** as, for instance, after severe typhoid fever. Among chronic infectious diseases, syphilis particularly is well known as a cause for neurasthenia. Not rarely **chronic diseases** generally, but particularly such as are attended with wasting discharges, are causes of neurasthenia. Enteroptosis also is a frequent cause of neurasthenia. Nor rarely the **too frequent bearing of children** and **unduly protracted lactation** are followed by neurasthenia. Neurasthenia is a disorder that at the present day is progressively increasing in frequency, because the demands upon the mental powers are constantly growing larger, and particularly because the demands are often grossly disproportionate to the time in which the work is to be done. The more restless and more active the life in a city and in a country, the greater is the number of neurasthenics likely to be. The city contains more neurasthenics than the country. Neurasthenia is especially prevalent in America, so that the disorder has even been designated the American disease. *Men* are more frequently neurasthenic than women, because they take a more prominent place in the arduous and heated struggle for existence. The disease is not unknown in *childhood,* and particularly schools for higher education under the direction of rigorous instructors are fruitful sources for the disorder.

Symptoms.—The symptoms of neurasthenia occasionally set in suddenly, or they may develop rather gradually, increasing progressively in intensity and number. Not rarely nervous disturbances referable to a single organ become so prominent that on superficial investigation there may be danger of assuming the existence of local visceral disease, and overlooking the neurasthenic cause. Among the most constant symptoms are **cerebral disturbances** (cerebrasthenia). The patients complain with especial frequency of undue readiness of fatigue from mental occupation, so that a sense of fulness, of pressure, and of beating in the head and headache, appear; while their thoughts wander and they are no longer able to follow the subject in hand. They may read for a considerable time without a knowledge of what they have read, for their thoughts have gone off in other directions. The sense of fatigue becomes progressively more troublesome, and gradually appears also after a brief period of mental activity. The patients are often harassed by the fear that mental impairment has set in, and often become so disheartened that they abstain from all mental occupation or on attempts to engage therein are seized with fear, excitement, and sweating. **Sleep** generally is greatly disturbed. The patients fall asleep only with much difficulty, continue to sleep for but a few hours, and then throw themselves about in bed for hours until day dawns. At the same time sleep is disturbed by restless dreams. The patient arises from bed in the morning with a sense of bodily **fatigue** and drags himself about throughout the day, without obtaining rest and recuperation on the following night. A sense of internal restlessness, and distracting thought and anxiety, prevent the much desired sleep. **A sense of fulness and of pressure in the head** at times persists continuously. Often **vertigo** is superadded to the other symptoms.

Not rarely neurasthenics are seized with **hallucinations** and **states of fear,** particularly the fear of traversing large spaces (fear of places, agoraphobia), of being within closed spaces (theaters, concert-halls), of riding in railway-cars, of standing on the banks of the river, etc. **Psychopathic states** develop but rarely, as, for instance, progressive paralysis of the insane. Frequently **spinal symptoms** appear, and many patients complain especially of pain in the course of the entire spinal column or at circumscribed areas thereof, although painful points cannot always be demonstrated objectively on percussion or on pressure. This condition was formerly designated *spinal irritation.* Sensations of burning and of beating not rarely appear in the sacral region. Some patients complain of a **girdle-sensation** or of a **band-like feeling** about the trunk. The symptoms described lead many patients to believe themselves suffering from a disease of the spinal cord, and especially tabes dorsalis, a misconception that becomes strengthened when paresthesias appear in the arms and the legs, or nervous disturbances in the activity of the badder and in the functions of the generative organs have developed. It is noteworthy that **inequality of the pupils** occurs in neurasthenic patients, although the pupils always react to the stimulation of light, and generally even more actively than in conditions of health. The **knee-jerks** also are generally not only preserved, but even increased. Nevertheless, they may be enfeebled or even abolished in some cases of neurasthenia. Neurasthenia generally occurs in emaciated, pallid individuals, whose general demeanor exhibits a certain degree of restlessness and irritability, and who furnish the physician with a detailed account of a long series of complaints, together with a report of subordinate and irrelevant conditions. **Vasomotor and secretory disorders** are not uncommon in cases of neurasthenia. Slight pallor and redness, and rapid alternation in these two conditions, occur with extreme frequency. There is a distinct tendency to *sweating;* the palms of the hands particularly are generally covered with cold sweat. Often derangement in the secretion of the *gastric juice* occurs; the patients not rarely suffer from *hypersecretion* and *hyperchlorhydria.* Disturbances in the *secretion of urine* may similarly occur, with particular frequency polyuria, the urine being conspicuous on account of its light color. and low specific gravity.

Taking up for consideration the **nervous disturbances in individual organs,** it will be found that among the *organs of special sense* the ear suffers with especial frequency, and the patient complains of roaring or ringing in the ears, often also of unilateral or bilateral impairment of hearing. The *eye* also is frequently involved. The appearance of spots and of flashes before the eyes and undue readiness of fatigue in reading are not uncommon complaints. Disturbances in the sense of smell and of taste are not often present. The patients are frequently annoyed by *paresthesias,* especially formication, a sense of cold, shooting pain, burning, and the like. These exhibit the widest variations with regard to distribution, intensity, duration, and recurrence. *Anesthesia,* but especially *hyperesthesia of the skin,* is likewise a well-known manifestation of neurasthenia, and it may be of indiscriminate distribution. The patients often complain of *muscular pains* and *undue readiness of fatigue in walking.* At times, also, the gait is unsteady and staggering. Some patients suffer so much pain in walking that they remain in bed for years—*akinesis algera;* or they collapse at once in standing or walking, although the strength of the legs in the recumbent posture is preserved—*astasia* and *abasia.*

Neurasthenics often suffer from total *loss of appetite* (*anorexia*), although in some, on the other hand, the *appetite is excessive* (*bulimia*). The patients frequently lose the sense of satiety. Often the ingestion of food is attended with disagreeable sensations, which persist throughout the period of gastric digestion, and give rise to the clinical picture of *nervous dyspepsia.* Under such conditions disagreeable and heavy sensations appear in the epigastrium, with a sense of pressure or even pain in the stomach. The patient complains of a sense of epigastric distention, and eructation is frequent. The face becomes markedly reddened, and there are increased flow of blood to the brain, a sense of beating in the head, and vertigo, a sense of fear, palpitation of the heart, and the like. Not rarely peristaltic unrest of the stomach, hyperchlorhydria, and hypersecretion, as well as gastroxynsis, appear in cases of neurasthenia. The *functions of the intestine* also are often deranged, as manifested by borborygmi, gaseous distention, and irregularity in the movement of the bowels. Occasionally the symptoms of *membranous enteritis* are encountered.

Often *cardiac symptoms* appear, especially subjective or even objective palpitation of the heart, and anginal attacks. The *pulse* is generally small, occasionally irregular, and frequently variable in rate. Occasionally, neurasthenic patients complain of *laryngeal symptoms*, such as a tendency to cough, a sense of tickling and of pain in speaking; and at times they suffer from spastic aphonia. Conditions resembling asthma also occur from time to time. The urine often contains an excess of *phosphates*. In many patients there is increased frequency of micturition in consequence of hyperesthesia of the bladder. In others there is difficulty in the expulsion of urine, on account of weakness of the detrusor of the bladder or of spastic contraction of the sphincter of the bladder. Incontinence of urine also may result from paralysis of the sphincter of the bladder. Often derangements in the *functions of the sexual organs* are present. Some patients exhibit abnormal sexual excitement, while coitus is followed by a sense of profound exhaustion. Complaint is often made of excessive seminal emissions. In other instances there is loss of sexual desire. Psychic impotence, spermatorrhea, and prostatorrhea may also readily develop.

Diagnosis.—The recognition of neurasthenia is not difficult. The neurotic general condition and the absence of anatomic alterations are especially to be taken into consideration.

Prognosis.—Neurasthenia is a most troublesome disorder, although it is unattended with danger to life. The prospects of cure are slight, for although material improvement, or even recovery can be brought about by physical and mental rest, particularly at the beginning of the disorder, such a result is not likely to be permanent, and the host of symptoms returns as before as soon as the patient resumes his former mode of life. Further, complete cessation of the disturbing occupation is not a certain remedy. On the contrary, some patients become the more greatly depressed by unpleasant thoughts when wholly unoccupied, and these may make the neurasthenia more pronounced than before.

Treatment.—Naturally in the treatment of neurasthenia consideration should first be given to the causative factors, and **causal therapy** should be instituted. The patient should especially avoid excessive mental activity, and should not continue at work for too long a time consecutively. He should, further, be cautioned against working at night. The use of wine, beer, coffee, tea, and tobacco should be interdicted, and a nutritious but easily digestible diet prescribed. It is advisable for the patient to give up his occupation for a considerable length of time, and seek rest and mental and physical recuperation in the mountains or the country, at the seaside, or in a protracted sea-voyage. Some of my patients have spoken highly of the beneficial influence of a trip along the Nile. Courses of **treatment with cold water** also are not rarely attended with good results. Not much is to be expected from **medicinal treatment.** Nervines have been employed, among which the bromids, arsenic, valerian, and strychnin are to be preferred. Greatly emaciated individuals are frequently improved by a course of **over-feeding. Electricity** and courses of **treatment at the springs** are frequently badly borne by neurasthenic patients. Successful results are but rarely obtained from **hypnosis** and **suggestion.** Recently **organotherapy** has been employed. Improvement is reported to have resulted especially from injections of testicular fluid, spermin, and sheep's brain.

TRAUMATIC NEUROSES.

Etiology.—The designation traumatic neuroses is applied to disturbances in nerve-function unattended with anatomic alterations in the nervous system, and resulting in the sequence of traumatism. The first cases were observed after *railway-accidents,* but a traumatic neurosis may just as readily develop after any other accident. At times the condition is attended with concussion of the entire body, while at other times only individual portions of the body are affected by the accident. Signs of external injury may be wholly wanting. It is noteworthy that not only physical, but also **mental shock** may be the cause for traumatic neuroses. Thus such a condition has been observed to develop in locomotive-engineers who have considered collisions between railway-trains unavoidable, but who have succeeded in averting them at the last moment. Often violent natural phenomena, such as an earthquake or a lightning-stroke in the vicinity, act as causes for traumatic neuroses. Traumatic neuroses are encountered with extreme frequency at the present time. In the first place, the extensive use of machinery in modern life is a most prolific source for causes of most varied kind; and, in addition, the laws relating to damages cause those interested to make special inquiry into the symptoms of the traumatic neuroses. There is, however, a certain justification for the statement with regard to accident-insurance that traumatic neuroses are imaginary diseases, as they are said to occur but rarely in countries in which the injured person is not entitled to damages. Nevertheless, hallucinations play an important part in the development of traumatic neuroses, and, without doubt, mental infection may occur among the injured. The disorder occurs principally in *adults* and in *men,* who are more commonly exposed to the risk of accident than are children and women. The traumatic neuroses develop more readily in *alcoholics* and in *syphilitic epileptics* than in those previously healthy.

Symptoms.—The symptoms of a traumatic neurosis may follow immediately upon an antecedent traumatism, or days, weeks, and even months of unimpaired health may elapse before nervous symptoms make their appearance. It is especially cases of the latter variety that often require a medico-legal opinion. **Cerebral disorders** are exceedingly frequent. Patients often complain of headache, a sense of pressure in the head, and vertigo, and these may be constantly present or appear paroxysmally. The patient readily becomes fatigued from mental exertion, and often exhibits apathy and indifference. Sleep is interfered with, and often disturbed by unpleasant dreams, and the memory becomes impaired. In some patients dementia and marked mental derangement develop. Some patients complain of peculiar sensations referred to the interior of the skull; as, for instance, that of a rolling or a dropping body. Also, severe pain at the vertex of the head is occasionally complained of, and this may resemble the hysterical clavus already described. Occasionally the patient suffers from **states of fear,** with especial frequency the fear of places—agoraphobia. The **temperament** almost always undergoes change. The patients become fearful, peevish, and capricious, are abnormally concerned about their condition, and are harassed with especial frequency by the fear of losing a suit for damages, or of the damages granted being too small. The patient is injuriously influenced especially by the circumstance that he may be considered ill by one physician and be declared a malingerer by another. Such patients often go from one physician to another, from one hospital to another, from one attorney to another, and become extremely contentious.

Spinal symptoms are manifested with especial frequency in abnormal sensations referred to the vertebral column. Impaired mobility and a sense of rigidity in the vertebral column, painful points on pressure, and a sense of burning in the situation of certain portions of the spinal column are frequently complained of, but a girdle-sense and a band-like feeling about the trunk also occur. Many patients complain of uncertainty in **gait.** They walk with the legs held far apart, swaying to and fro, and become readily fatigued. **Tremor** not rarely sets in, and this may be confined to but a single member or it may involve the entire body. **Paralysis** and **contractures** in monoplegic, paraplegic, and hemiplegic distribution have been observed. In the presence of hemiplegia the cerebral nerves, especially the facial and the hypoglossal, remain uninvolved. It is often noteworthy that numerous muscles on percussion and on exposure exhibit active **fascicular twitching.** The **knee-jerks** are frequently increased, although in rare instances they are wanting. **Paresthesiæ** in the lower extremities are often complained of. **Hyperesthesia** and **anesthesia** also occur in most varied distribution and extent.

The **eye** and the **ear** are not infrequently affected. The pupils not rarely are unequal, and in exceptional instances the reaction to light-stimulation is wanting. Contraction of the visual fields, however, is of especial importance. The patient often complains of roaring and ringing in the ears, and occasionally there is unilateral or bilateral impairment of hearing. Some patients suffer from subjective or objective **palpitation of the heart.** Frequently the pulse becomes considerably accelerated on slight mental or physical exertion, or on pressure upon certain parts of the body. Asthmatoid states also occur occasionally. Some patients complain of **vesical disorders,** at times of retention of urine, at other times of incontinence. Occasionally hyperesthesia of the bladder is present, and is attended with increased frequency of micturition. **Alimentary glycosuria** has been observed in a number of instances; less commonly, **diabetes mellitus.** At times derangement in the functions of the **generative organs** occurs: excess or absence of seminal emissions or of sexual desire and impotence.

The *duration of traumatic neuroses* is susceptible of great variations. An especially favorable influence is often exerted by the awarding of pecuniary indemnity for an accident received, and some patients become thereafter perfectly well and capable of resuming their occupation. It is especially such occurrences that frequently cause judges and accident-insurance officials to consider all of the previous symptoms as malingering, a view that is often incorrect, for it may be asked why should not morbid mental processes disappear when the mind can continue to operate under more favorable conditions. Not a few victims of accidents have been accused of malingering, and have eventually wound up in hospitals for the insane. There can be no doubt that error in this connection has often been committed by the attending physician and by the judge, whose opinion is based upon that of the former.

Diagnosis.—The diagnosis of traumatic neurosis is not rarely a most difficult problem. It is, in the first place, important to have a knowledge of a physical or a mental shock, and in this connection not so much is dependent upon the severity of the accident as upon the intensity of the mental impression. Without doubt there are persons who not rarely simulate the clinical picture of a traumatic neurosis, which they have learned from reports of accidents. It may also happen that they have been instructed in the methods for securing damages by patients who have been successful in attaining this result. It should not be overlooked that the victims of accidents are likely to exhibit a marked tendency to exaggerate their symptoms, and that under such circumstances it is difficult to distinguish the actual from the exaggerated. In forming a medico-legal opinion with regard to a traumatic neurosis it is of especial importance to demonstrate **objective alterations,** as, for instance, contraction of the visual field, tachycardia on pressure upon certain parts of the body or after slight physical exertion, and the like. It has been maintained by some physicians, not without reason, that no such condition as the traumatic neuroses exists, but that all of the symptoms are manifestations of traumatic neurasthenia or hysteria, or of both diseases together; but it is, nevertheless, not without practical value to adhere to the conception of the traumatic neuroses.

Prognosis.—The traumatic neuroses terminate fatally with comparative rarity, but they greatly impair the capacity of the patient for work, and occasionally they persist throughout the whole of life if the patient has entered suit for pecuniary damages or lives in poor circumstances. The prognosis must, therefore, be made with great reserve as regards complete recovery.

Treatment.—The treatment must, above all, be psychic. Little can be accomplished with drugs, of which **nervines**, and especially bromids, may be employed. Removal from the scene of the accident and a **sojourn amid new surroundings** are often useful. Occasionally **hypnosis** and **suggestion** are helpful.

PART VI.

DISEASES OF THE MUSCLES.

PROGRESSIVE MYOPATHIC MUSCULAR ATROPHY.

Etiology.—The motor-trophic ganglion-cells in the anterior horns of the spinal cord, the motor nerve-fibers arising from them, and the related muscular fibers represent a continuous, uninterrupted whole. Disease of the ganglion-cells of the anterior horns is responsible for the spinal progressive muscular atrophy previously described; disease of the peripheral motor nerves gives rise to neural progressive muscular atrophy; and, finally, atrophic processes of progressive character may result in consequence of disease of the muscles themselves. Cases of the last-named variety are included under the designation myopathic muscular atrophy, for which the name progressive muscular dystrophy also has been proposed. In contradistinction from spinal progressive muscular atrophy, myopathic muscular atrophy is a markedly *hereditary* and *familial* disorder, which may be found in some families, particularly among the males, through many generations. This fact seems to indicate that the disease is frequently the result of a morbid predisposition residing in the muscles. Occasionally the disorder has been observed to develop in the sequence of antecedent **infectious diseases.** **Exposure to cold** and **traumatism** also are included among the causal factors. The disease occurs most commonly in *males.* It often begins in *childhood,* and generally before the twenty-fifth year of life.

Symptoms.—Several types of myopathic progressive muscular atrophy have been distinguished accordingly as the disorder has begun in one or another portion of the body, and has given rise to corresponding deformities. We shall confine ourselves to a description of three principal varieties, which may be designated pseudomuscular hypertrophy, juvenile and infantile muscular atrophy.

PSEUDOMUSCULAR HYPERTROPHY.

Some children present symptoms of pseudomuscular hypertrophy from birth, while in others the symptoms appear before the end of the second year of life, and rarely even later. The disease can be recognized from the fact that the patients learn to walk late and imperfectly, and that they are easily fatigued in walking. The *muscles of the calf* generally are greatly increased in size, so that they have been designated athletic or herculean muscles are incapable of any considerable degree of activity. The *extensors of the thighs* and the *gluteal muscles* also present evidences of pseudohypertrophy. The remaining muscles of the legs, however, the muscles of the back, and also those of the arms are thin and atrophic. In the dorsal decubitus the *feet* occupy the position of pes varo-equinus. In walking the patient raises the feet high in order to avoid striking the ground with the toes, and the gait becomes uncertain and waddling. *Atrophy and weakness of the muscles of the back* give rise in the standing posture to lordosis in the lumbar region, and kyphosis in the dorsal region of the spinal column. On attempting to sit down the patient rather drops, and if he be requested to arise from a sitting position upon the floor to the erect posture, he will first make twisting and turning movements with the upper part of the body; then he will support himself upon his arms, then rest upon the knees, and then climb upon his legs and thighs with his hands. The muscles of the face generally remain uninvolved. The *mechanical* and the *electric irritability* of the affected muscles is diminished quantitatively in accordance with the degree of atrophy. Indications of degenerative electric reaction do not appear. *Fascicular muscular twitching* is not observed. The *layer of subcutaneous fat* generally is unusually thick over the pseudohypertrophic muscles. The *skin* is, as a rule, cyanotic, bluish, and marbled, and feels cold. The *knee-jerk* is diminished in consequence of involvement of the extensor of the thigh, or it may be wholly abolished. The *duration of the disorder* may extend over many years. Under such circumstances *muscular contractures* not rarely develop. Death results from some accidental disease or from excessive exhaustion.

JUVENILE MUSCULAR ATROPHY.

Juvenile muscular atrophy is, in the first place, attended with wasting and resulting weakness in the *muscles of the shoulder-girdle and the chest*. The greater and the lesser pectoral, the greater anterior serratus, the rhomboid, the trapezius, and the latissimus dorsi especially undergo atrophy. In consequence of paralysis of the serratus the inner borders of the shoulder-blades stand off from the back (p. 156, Fig. 29), and if the arms are elevated from below the elbows, the scapulæ are readily displaced upward, so that the head sinks to a certain degree between them. The scapulæ thus exhibit an excessive range of displacement and of movement. Wasting and weakness of the *muscles of the back* give rise in the standing posture to lordosis in the lumbar region and kyphosis in the dorsal region of the vertebral column. They render extremely difficult change from the recumbent to the erect posture. In the *upper arm*, especially the biceps, the brachialis internus, the long supinator, and, at a later period, also the triceps, undergo atrophy, while of the muscles of the forearm the extensors become involved. The flexors and the small muscles of the hand, however, remain unaffected. In the *lower extremities*, which generally are involved much later than the arms, the glutei, the quadriceps of the thigh, and the tensor of the fascia lata, and in the leg the anterior tibial, undergo atrophy. Naturally, the power of walking is as a result greatly disturbed. In walking the patient lifts the legs high, and the gait is waddling. In addition to the muscular atrophy *hypertrophy* of other muscles takes place, particularly in the deltoid, the supraspinatus, the infraspinatus, and the muscles of the calf. In contrast with the wasted muscles of the upper arm, the deltoid region is conspicuous on account of its unusual fulness (Fig. 29),

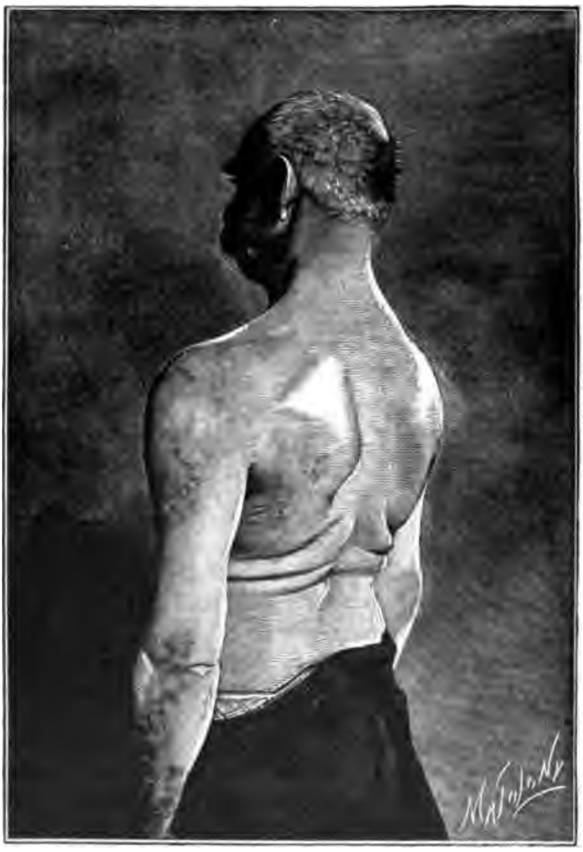


FIG. 29.—Juvenile muscular atrophy in a man 56 years old; from a photograph (personal observation, Zurich clinic).

and a similar disproportion exists between the atrophied muscles of the thigh and the gluteal muscles, on the one hand, and the hypertrophied muscles of the calf on the other hand. The affected muscles feel firm, do not exhibit fascicular contraction, but only simple quantitative diminution in electric irritability, and no reaction of degeneration. Sensory disturbances, as well as paralysis of the bladder and the rectum, do not occur. The muscular atrophy most commonly begins on both sides, but its commencement is not rarely so gradual that it cannot often be determined with certainty. The *duration of the disease* generally extends over many years. The patients become progressively more helpless, and death finally occurs in consequence of excessive exhaustion or of some intercurrent disease (pneumonia, pulmonary tuberculosis).

INFANTILE MUSCULAR ATROPHY.

The disease begins with wasting of some of the *muscles of the face*, particularly the sphincter of the eye and of the mouth. As a result the palpebral fissure is so wide that the eye appears about to bulge through it, while the lips remain open and form a proboscis-like protrusion. The forehead is smooth and free from wrinkles, and the entire facial expression acquires a stupid character. The appearance has therefore been designated *myopathic facies*. In laughing the extent to which the mouth is widened is conspicuous. Gradually, but occasionally only after the lapse of more than five years, the atrophy extends to the muscles of the shoulder-girdle, the arms, the back, and even the legs. Especially the greater and the lesser pectoral, the trapezius, the deltoid, the biceps, the internal brachial, the triceps, the long supinator, the radial extensor of the carpus, and the dorsal extensors are involved, and corresponding deformities and functional disturbances result in consequence. Muscular hypertrophy, however, does not occur. In other respects the conditions are much the same as in cases of pseudomuscular hypertrophy and juvenile muscular atrophy.

Diagnosis.—The recognition of myopathic muscular atrophy is not difficult, for in contradistinction from **spinal progressive muscular atrophy** the disorder generally begins in childhood, is an hereditary or familial disorder, and does not present either fascicular twitching in the hypertrophied muscles or degenerative electric reaction. It further does not involve the small muscles of the hands, which are most commonly attacked in cases of spinal muscular atrophy, and bulbar symptoms are never present. The absence of degenerative electric reaction distinguishes the disease also from **neural muscular atrophy** and from muscular atrophy in consequence of acute or chronic *polyneuritis*, as well as from **spinal paralysis of childhood.** The commencement and the distribution

while of
The fle
unaffect
much la
and th
underg

variety of myopathic mus-

from anatomic lesion
nerves is distinctive of all
The changes in the mus-
pseudomuscular hypertrophy.
plasia of the interfibrillary
muscular fibers progres-
become hypertrophied. The
characterized by marked trans-
of nuclear multiplication.
fatty degeneration, fibril-
ation, and vacuolation. An
may take place in the inter-
ently hypertrophied muscles.
the interstitial connective
while in cases of *infantile mus-*
fibers are not encountered, the
little increased, and develop-

ular atrophy is unattended with
: nevertheless the prognosis is
is helpless in the face of a dis-
tendency to advance, and finally
work and movement.
known, and also *internal remedies*
checking the progress of the dis-
The best results can be expected
pedic appliances.

SSIVE MUSCULAR ATROPHY.

ic Alterations.—Neural progressive
myopathic muscular atrophy, is usually
which generally begins in *early*
more commonly than girls. It depends
normal predisposition on the part of the
little is known with regard to the *anatomic*
ion and atrophy of the medullary sheaths of
are reported to have been found, while the
uninvolved, or at any rate has exhibited de-
the columns of Goll. An observation has,
tly reported in which the spinal cord and the
were found intact, and atrophy was demonstra-
ffected muscles.

and Diagnosis.—The disease begins insidiously,
of the peroneal, the common extensor of the

digits and the small muscles of the toes and the foot, so that club-foot develops, which does not, however, greatly annoy the patient. If atrophy and weakness in the anterior tibial and the long extensor of the great toe are superadded, the foot acquires the position of *pes varo-equinus*, and, in consequence, there occurs great difficulty in walking. Gradually the muscles of the calf and the thigh are involved in the wasting. The muscles of the trunk and the face remain uninvolved, while gradually, but occasionally only after the lapse of many years, the muscles of the ball of the hand and of the fingers and the extensors of the forearm are involved, and, eventually, also those of the upper arm and the shoulder. The atrophied muscles are often sensitive to pressure. They exhibit **fascicular twitching** and **partial** or **complete degenerative electric reaction. Mechanical irritability** is generally diminished. The **tendon-reflexes** are enfeebled or wanting in the distribution of the atrophied muscles. Often complaint is made of **paresthesia.** Occasionally, hyperesthesia is present, rarely anesthesia. The **bladder** and the **rectum** are not affected. The *disorder*, as a rule, involves symmetrical muscles simultaneously, and pursues a slow course. In contradistinction from **myopathic muscular atrophy,** it is characterized by degenerative electric reaction, and fascicular muscular twitching, and it is distinguished from **spinal progressive muscular atrophy** by the fact that it begins in the muscles of the toes and the foot, and is attended with sensory disturbances.

The **prognosis** and the **treatment** are the same as for myopathic muscular atrophy.

TRUE MUSCULAR HYPERTROPHY.

True muscular hypertrophy is an exceedingly *rare disorder*, which is characterized by hypertrophy of individual muscle-fibers, without alteration in the interstitial muscular tissue. The muscle-nuclei are generally enlarged and increased in number, and the transverse striation of the muscular fibers is quite marked. Among the *causative factors* typhoid fever, excessive muscular activity, and traumatism have been mentioned. The disorder is also reported as having been observed as a congenital condition. The disease occasionally affects but a few muscles, while in other instances symmetrical parts are involved. Under such conditions only the muscles of the extremities are affected, and not those of the trunk or the face, although enlargement of the tongue—macroglossia—has at times been described. The muscles are notable for their large size, although, as a rule, their functional power is impaired. The electric irritability is unaltered or slightly diminished, although the mechanical irritability, however, is at times increased. The disorder begins sometimes with paresthesiæ, and of the muscular wasting determine the *variety of myopathic muscular atrophy.*

Anatomic Alterations.—The freedom from anatomic lesion of the spinal cord and the peripheral nerves is distinctive of all varieties of myopathic muscular atrophy. The changes in the muscles have been best studied in cases of *pseudomuscular hypertrophy.* In this condition there is found hyperplasia of the interfibrillary connective tissue, while a portion of the muscular fibers progressively disappear and another portion become hypertrophied. The atrophied muscular fibers are often characterized by marked transverse striation. They are also the seat of nuclear multiplication. Some fibers exhibit granular turbidity, fatty degeneration, fibrillary degeneration, vitreous degeneration, and vacuolation. An abundant development of fat-cells may take place in the interstitial connective tissue of the apparently hypertrophied muscles. In cases of *juvenile muscular atrophy* the interstitial connective tissue is said to remain unaltered, while in cases of *infantile muscular atrophy* hypertrophied muscle-fibers are not encountered, the interstitial connective tissue is but little increased, and development of fat is wanting in the latter.

Prognosis.—Myopathic muscular atrophy is unattended with immediate and direct danger to life; nevertheless the prognosis is not favorable, because the clinician is helpless in the face of a disorder that exhibits an irrepressible tendency to advance, and finally renders the patient incapable of work and movement.

No *prophylactic measures* are known, and also *internal remedies* are wanting that are capable of checking the progress of the disease or even of effecting its cure. The best results can be expected from massage, baths, and orthopedic appliances.

NEURAL PROGRESSIVE MUSCULAR ATROPHY.

Etiology and Anatomic Alterations.—Neural progressive muscular atrophy, like myopathic muscular atrophy, is usually an *hereditary* or *familial disease,* which generally begins in *early childhood* and attacks *boys* more commonly than girls. It depends probably upon an abnormal predisposition on the part of the peripheral nerves. Little is known with regard to the *anatomic alterations.* Destruction and atrophy of the medullary sheaths of the peripheral nerves are reported to have been found, while the spinal cord has been uninvolved, or at any rate has exhibited degeneration only in the columns of Goll. An observation has, however, been recently reported in which the spinal cord and the peripheral nerves were found intact, and atrophy was demonstrable only in the affected muscles.

Symptoms and Diagnosis.—The disease begins insidiously, at first with atrophy of the peroneal, the common extensor of the digits and the small muscles of the toes and the foot, so that club-foot develops, which does not, however, greatly annoy the patient. If atrophy and weakness in the anterior tibial and the long extensor of the great toe are superadded, the foot acquires the position of *pes varo-equinus*, and, in consequence, there occurs great difficulty in walking. Gradually the muscles of the calf and the thigh are involved in the wasting. The muscles of the trunk and the face remain uninvolved, while gradually, but occasionally only after the lapse of many years, the muscles of the ball of the hand and of the fingers and the extensors of the forearm are involved, and, eventually, also those of the upper arm and the shoulder. The atrophied muscles are often sensitive to pressure. They exhibit **fascicular twitching** and **partial** or **complete degenerative electric reaction. Mechanical irritability** is generally diminished. The **tendon-reflexes** are enfeebled or wanting in the distribution of the atrophied muscles. Often complaint is made of **paresthesia.** Occasionally, hyperesthesia is present, rarely anesthesia. The **bladder** and the **rectum** are not affected. The *disorder*, as a rule, involves symmetrical muscles simultaneously, and pursues a slow course. In contradistinction from **myopathic muscular atrophy**, it is characterized by degenerative electric reaction, and fascicular muscular twitching, and it is distinguished from **spinal progressive muscular atrophy** by the fact that it begins in the muscles of the toes and the foot, and is attended with sensory disturbances.

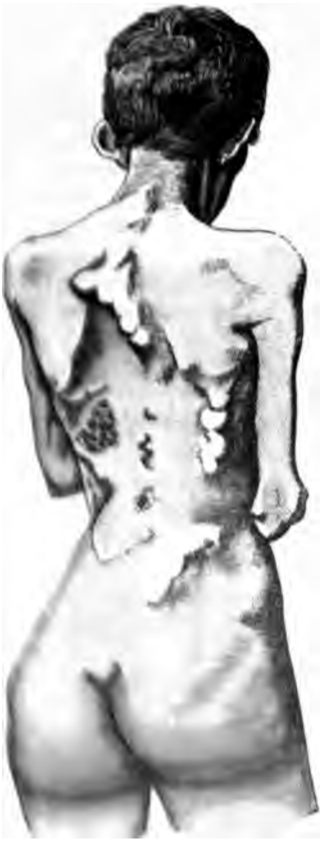
The **prognosis** and the **treatment** are the same as for myopathic muscular atrophy.

TRUE MUSCULAR HYPERTROPHY.

True muscular hypertrophy is an exceedingly *rare disorder*, which is characterized by hypertrophy of individual muscle-fibers, without alteration in the interstitial muscular tissue. The muscle-nuclei are generally enlarged and increased in number, and the transverse striation of the muscular fibers is quite marked. Among the *causative factors* typhoid fever, excessive muscular activity, and traumatism have been mentioned. The disorder is also reported as having been observed as a congenital condition. The disease occasionally affects but a few muscles, while in other instances symmetrical parts are involved. Under such conditions only the muscles of the extremities are affected, and not those of the trunk or the face, although enlargement of the tongue—macroglossia—has at times been described. The muscles are notable for their large size, although, as a rule, their functional power is impaired. The electric irritability is unaltered or slightly diminished, although the mechanical irritability, however, is at times increased. The disorder begins sometimes with paresthesiæ, and is attended with vasomotor disturbances. The *prognosis* and the *treatment* are the same as those for myopathic muscular atrophy.

PROGRESSIVE OSSIFYING MYOSITIS.

Progressive ossification of the muscles is an *exceedingly rare disease*, which generally begins *before the fifteenth year of life* and occurs *more frequently in males* than in females. Nothing of a definite nature is known with regard to its etiology, although exposure to cold and traumatism, as well as diseases of the spinal cord, have been mentioned as causative factors. As a rule, the *muscles of the back and the nucha* are involved earliest, then those of the neck and the chest, and finally also the muscles of the arm, the legs, and of mastication, some of the muscles of the face, rarely those of the soft palate; while the abdominal and the laryngeal muscles, the sphincters of the bladder and the rectum, the heart, the diaphragm, the tongue, and even the small muscles of the hand remain uninvolved. The process of ossification advances with extreme slowness, and the disease is often protracted for more than twenty years. Either one muscle after another is gradually involved or symmetrical muscles are affected almost simultaneously. The changes in the muscles not rarely begin with *fever* and *severe local pain*. The skin over the painful area is reddened and edematous. As the cutaneous edema recedes an area of doughy resistance becomes appreciable in the subjacent muscle; this area disappears only in rare instances and generally feels firmer, and grows gradually denser and [illegible] eventually becoming as hard as bone. Obviously connec- [illegible] inflammation has first taken place, which slowly undergoes [illegible] into cartilage, and then ossification, a process that [illegible] recognized and studied from the sense of resistance



[illegible] ance of the back [illegible] ossifying myo- [illegible] years old (after [illegible] that is appreciated by means of puncture with a needle. The bon masses have at times a rather plate-like form, while at other time they are band-like, and at still other times they resemble processe with numerous sharp teeth. Often the ossified muscles form adhesions to the subjacent bone, and in consequence become immobile The bones themselves readily become the seat of exostoses. Th larger the number of muscles involved in the ossifying process, th more rigid and immobile does the patient grow. He become incapable of moving the vertebral column, the neck, the head ossification of the muscles of the chest gives rise to dyspnea, tha of the pectoral causes the arm to be constantly adducted to th chest; ossification of the biceps gives rise to flexion and immobility of the forearm, while involvement of the muscles of mastication interferes with the ingestion of food. Eventually th patient becomes as rigid as a stick, and death results in consequence of progressive exhaustion. Not rarely patients hav exhibited simultaneously *deficient development* (*shortening*) *of th great toe*, and less commonly of the thumb. The disease i readily recognizable, for not only can the ossification be easil detected on palpation, but it is not rarely even appreciable to th eye (Fig. 30). The ossifying process arises from the tendinou attachments and extends to the muscular tissue.

The **prognosis** is unfavorable with regard to cure.

Effective **treatment** is as yet unknown. The best result may be expected from warm **baths** and **massage**.

POLYMYOSITIS.

Multiple inflammation of the muscles is probably an *infectiou disease*, which occurs either as an independent disorder or as sequel of other infectious diseases, as, for instance, puerperal fever In the manner of an infectious disease, the disorder frequently set in with *chill* and *fever*, together with demonstrable *enlargement o the spleen*. The patient often complains of pain in the joints which are swollen. Also, the skin is edematous, and the subjacen muscles feel thickened and doughy, and are exceedingly tender o pressure. The electric irritability is diminished. The tendon reflexes are enfeebled or wholly abolished. Pressure upon th nerve-trunks is unattended with pain. Not rarely measly, scarlatiniform, and herpetic eruptions appear upon the skin, so tha the disease has been designated also dermatomyositis. Should th cutaneous edema subside, the muscles become denser and dense and contractured. The forearm becomes flexed at an acute angl with especial frequency, so that the appearance is suggestive of th presence of trichiniasis. The disease is generally attended wit severe pain, often also with albuminuria. Frequently it terminates fatally within from two to four weeks, amid progressiv exhaustion. Less commonly it pursues a chronic course and terminates in recovery. Excised bits of muscle present a light color, and appear as if watered and swollen, while microscopic examination discloses granular turbidity, fatty degeneration, vitreous degeneration, and vacuolation in the muscle-fibers, together with hyperplasia of the interstitial connective tissue. The brain, the spinal cord, and the peripheral nerves are unaltered.

In **treatment** **warm baths, massage, salicylic acid, and sodium salicylate,** as well as **faradization** of the muscles, have been employed.

MYOTONIA.

Myotonia is known also as *Thomsen's disease*, and is generally an hereditary affection. In isolated instances it has been observed to develop in the sequence of *fright* and *over-exertion.* The disease has not rarely been seen in the newborn, while in other instances the earliest symptoms have not appeared until childhood, and occasionally not before the twentieth year. The essential feature of the disease consists in the circumstance that the muscles after every contraction remain for some time in a state of tonic, painless contraction, so that an intended movement is prevented or is executed most incompletely. Should the patient desire to stand up and to walk, elevation of the body is effected only with great difficulty and slowness, while often the patient falls and rolls to and fro upon the ground, and only after some time does walking become possible. If an object be grasped, it cannot at once be released, as, for instance, in taking the hand of the physician. The muscles of the face and the tongue are not rarely involved, and then the speech becomes drawling and halting. Even the ocular muscles may be affected. The disorder generally begins in the lower extremities and gradually extends upward. The sphincters of the bladder and the rectum remain uninvolved. The affected muscles generally become increased in *size*, but are nevertheless impaired in function. Occasionally they exhibit *fascicular contraction.* The muscles at times are also tender on pressure. The *myotonic reaction* is a notable feature. If the muscles are stimulated with a strong galvanic or faradic current, the muscular contractions persist for from twenty to thirty seconds beyond the stimulation. On stimulation with a number of opening faradic currents, however, short and lightning-like muscular contractions take place. On stimulation with the galvanic current, contractions are obtained only on closure. If one pole of the galvanic current is placed in the palm of the hand, and the other upon the nape of the neck, rhythmic muscular waves will soon be observed in the flexors of the fingers, passing from the kathode to the anode. Quantitative alterations in electric irritability are not known to occur. Electric stimu' the related motor *nerves* also discloses no quantitative changes. The nerves likewise exhibit no qualitative alteration, except that on labile application of the galvanic current tonic contractions appear in the related muscles and persist for as long as ten minutes afterward. The *mechanical* like the electric *irritability of the muscles* also induces a persistence of the contraction. The *tendon-reflexes* are at times unaltered, at other times exaggerated, and at still other times enfeebled. *Cutaneous sensibility* is unaltered, although some patients complain of paresthesia. The disorder generally persists throughout life; only rarely has recovery been observed. The symptoms are usually aggravated by cold, while the movements are more readily executed after repeated attempts. The brain, the spinal cord, and the peripheral nerves have been found unaltered on postmortem examination, while increased width, granular clouding, vitreous degeneration, vacuolation, increase in the muscle-nuclei, and degeneration, together with empty sarcolemma-sheaths, with slight hyperplasia of the interstitial connective tissue, have been demonstrated in the *muscle-fibers*. Myotonia is not dangerous to life, but is a most troublesome disorder, preventing patients from the pursuit of their occupations and rendering them incapable of military service. Subjective relief is occasionally afforded by means of *massage* and *tepid baths*.

MYASTHENIA.

Myasthenia consists in an undue readiness of fatigue on the part of the muscles. If the patient raises his arms aloft several times, progressive weakness becomes manifest, and the arms appear for a time incapable of movement, as if paralyzed. A like condition occurs also in the legs. Even the muscles of speech and of deglutition become exhausted rapidly, and eating is attended with the danger that a bolus of food may become impacted and cause death by suffocation. The muscles of the eyelids also readily become exhausted; the eyelids close and cannot be opened. Profound involvement of the bulbar nerves gives rise to a condition closely resembling chronic bulbar paralysis, except that the muscles recover within a short time and occasionally manifest their functional activity. If the muscles be stimulated electrically, they likewise soon become exhausted, and no longer respond.

Nothing of a definite nature is known with regard to the **etiology** of this rare disease, although of late the possibility of **auto-intoxication** has been considered.

The **treatment** should be directed to invigorating the body by means of a nutritious **diet** and of **rest**. In addition, preparations of **arsenic, cinchona,** and **iron** have been recommended.

PART VII.

DISEASES OF THE SKIN.

I. INFLAMMATIONS OF THE SKIN (DERMATITIDES).

ERYTHEMATOUS INFLAMMATIONS OF THE SKIN (ERYTHEMATOUS DERMATITIDES).

URTICARIA (NETTLE-RASH; HIVES).

Symptoms and Diagnosis.—Urticaria is characterized by the formation of **wheals or pomphi,** which are often attended with intolerable **itching.** The wheals occur especially upon the **face** and the **trunk,** and less commonly upon the extremities. At times but a few disseminated wheals are present, while at other times the body is densely covered with them, so that often adjacent wheals coalesce (urticaria conferta) and form at the same time irregular bands (gyrate urticaria). The wheals vary in **size** from that of a lentil to that of the palm of a hand, and even beyond. The designation giant urticaria also is applied to wheals of unusually large size. The wheals are at times uniformly red in **color,** while at other times they are pale at the center, and only the elevated border is red (urticaria porcellanea).

In rare instances vesicles or blebs form upon the wheals (*miliary, vesicular,* or *bullous urticaria*). Occasionally wheals occur also upon the *mucous membranes,* as, for instance, that of the cheeks, the pharynx, the larynx, and the bronchi.

The *duration of the individual wheals* is extremely variable. Not rarely they disappear within a short time, while new wheals appear at other points upon the skin. Occasionally the disorder is confined to the local alterations in the skin, and it disappears in the course of a few hours—*ephemeral* or *evanescent urticaria.* In other instances it begins with chilliness and fever, or fever soon develops after the appearance of the wheals—*nettle-fever, febris urticata;* but recovery generally takes place in the course of a few days. There is, further, a chronic variety of urticaria—*urticati-*

catio, *chronic urticaria*, *urticaria perstans*—the disease persisting for months or years, or even throughout the whole of life, less commonly without intermission, but rather in constantly repeated recurrences. The patients are greatly distressed by the intense itching by day, and particularly by night, and they are gradually reduced to a state of alarming exhaustion.

A chronic variety of urticaria has been designated *pigmented urticaria*, beginning in the first months of life, persisting to the tenth year, and attended with the presence of reddish or brownish, slightly elevated pigment-areas upon the skin, instead of wheals.

Anatomic Alterations.—The anatomic alterations in the skin must be studied during life, as the manifestations largely disappear after death. If a wheal be punctured and be compressed from the side, a small amount of clear fluid is expressed, and the wheal collapses. The histologic alterations in the skin have been studied especially in wheals induced artificially in animals. Dilatation of the blood-vessels and the lymphatics of the cutis was found, although the blood-vessels may be compressed, narrowed, and emptied, in consequence of edema of the cutis. Colorless blood-corpuscles and mast-cells had accumulated upon the outer aspect of the vessels. The cutis was edematous, and occasionally swelling could be demonstrated also in the lowermost epithelial cells of the epidermis.

Etiology.—Urticaria is most commonly of toxic origin, while mechanical and nervous influences are less commonly operative. **Toxic causes** occasionally exert their influence from without directly upon the skin, and it is well known that contact with the stinging nettle, Rhus toxicodendron, and certain caterpillars, the external application of carbolic acid and of poultices may give rise to urticaria, as well as the bite of a number of insects (flea, mosquito, bedbug) or of the leech. In other instances toxic substances reach the skin only through the intermediation of the blood-vessels and the lymphatics. Urticaria is therefore not an uncommon manifestation in connection with gastric and intestinal catarrh, as well as with jaundice. Some individuals are always attacked with urticaria after the ingestion of certain articles of food (strawberries, lobster, crabs, cheese, oysters) or of certain drugs (quinin, salicylic acid, antipyrin, balsamics, potassium iodid, etc.). Urticaria occurs not rarely after puncture of echinococcous or ovarian cysts, obviously because in the act of puncture some of the cystic fluid has found its way into the abdominal cavity and by absorption has gained entrance into the general circulation. Urticaria occurs occasionally also in association with intestinal worms. In this connection the cases should be considered that develop after infectious diseases (relapsing fever, typhoid fever, scarlet fever, measles, pulmonary tuberculosis) and after diseases attended with profound disturbances in metabolism (diabetes, nephritis, carcinoma).

The **mechanical causes** for urticaria include those that induce active scratching of the skin. In the presence of cutaneous parasites, the mechanical effect is often superadded to the toxic effect. Itching diseases of the skin (prurigo, pruritus) also are generally followed by urticaria. Urticaria develops in some individuals when the skin has been exposed to the action of the wind. In some persons, particularly neurotic and hysterical individuals, the skin is so sensitive that urticaria can be induced in any desired design by means of a hard body—*factitious* or *dermographic urticaria.* Especially **neurotic influences** can be readily discovered in some cases of urticaria. The affection occurs frequently in association with hysteria, occasionally with neuralgia, and it may develop also after fright and joy, or through reflex influences in connection with dentition and uterine disorders.

Urticaria has been designated an *angioneurosis* of the skin.

Prognosis.—Although urticaria is not a dangerous disorder, it may in its chronic form become extremely troublesome, and drive the patient almost to distraction.

Treatment.—The **prophylaxis** should be directed to the avoidance of food and drugs that experience has shown to be capable of inducing urticaria. **Causal treatment** should be directed to the removal of causative conditions (destruction of insects, relief of gastro-intestinal catarrh, etc.). **Symptomatically,** inunctions of carbolized ointment (1 : 10) or affusions of a solution of carbolic acid (5.0 : 100), or the local application of lotions containing *lemon-juice*, *vinegar*, or *cologne-water*, may be recommended. Internally, *salol* (salol, 1.0—15 grains; saccharin, 0.02—$\frac{1}{3}$ grain; 1 powder every two hours) may be administered. Occasionally successful results have been obtained from the employment of *potassium iodid*, *quinin*, *carbolic acid*, *arsenic*, *atropin*, *potassium bromid*, or *pilocarpin*. The use of tepid baths also appears worthy of recommendation. The patients should wear soft and warm *underclothing*, and avoid thick *feather-beds*.

NODOSE ERYTHEMA.

Symptoms and Diagnosis.—Nodose erythema begins not rarely in the manner of an acute infectious disease, with **chill and fever**. The patient feels extremely languid, and presents enlargement of the spleen. At the same time or in a short while **articular pains** appear, most commonly at the knee and the ankle. The joints are swollen, and the overlying skin often is reddened. The attention of the patient is generally directed to the skin by the burning and prickling sensations. There form **red nodules,**

covered by tense, smooth, and glistening skin, and feeling hot. The number of nodules varies widely, at times only a few being encountered, while at other times they are packed close together. The nodules vary in size from that of a pea to that of the palm of a hand. They appear earliest and generally in largest number upon the anterior aspect of the leg, while the arms are affected later, and occasionally remain wholly uninvolved. Nodules are rarely encountered upon the trunk, and scarcely ever upon the neck or the face.

In isolated instances nodules have been observed upon the *mucous membranes*, as, for instance, that of the mouth, the larynx, the vagina, and the conjunctiva.

The appearance of the nodules changes in the course of a few days. They become rather brownish-red, bluish, greenish, yellowish, and the patient often presents an appearance as if he had been struck in numerous places. The disorder has therefore been designated also *contusiform dermatitis*. This variation in color is due to the circumstance that diapedesis of red blood-corpuscles has taken place within the nodules, and the cells undergo gradual degeneration with corresponding changes in their hemoglobin.

Only in rare instances do *pustules* appear upon the nodules, so that the disorder may be confounded with small-pox. *Ulcerative destruction* of the nodules also occurs but exceptionally, and in consequence of which a resemblance to ulcerative cutaneous syphilis may result.

Recovery from the disease generally takes place within from two to four weeks, while less commonly the disorder is protracted for a longer period, with numerous recrudescences. *Complications* rarely occur, as, for instance, acute endocarditis. Some physicians maintain that pulmonary tuberculosis develops as a sequel of nodose erythema, although in spite of a not inconsiderable experience I have not yet been able to confirm this statement.

Anatomic Alterations.—Dilatation of the blood-vessels and of the lymphatics of the skin, and accumulation of round cells upon the outer aspect of the blood-vessels, together with edema of the cutis, have been found in the nodules.

Etiology.—Nodose erythema is, in all probability, an **infectious disease,** although it has not as yet been possible to discover a specific bacterium. The disorder may occur as a **primary** affection, or develop **secondarily** in the course of other infectious diseases, particularly after follicular angina, typhoid fever, pulmonary tuberculosis, scrofulosis, malaria, syphilis, and gonorrhea. Primary nodose erythema occurs in the spring and autumn occasionally in epidemic distribution and in association with erysipelas and herpes zoster. Women are somewhat more frequently attacked by the disease than men.

Prognosis.—The disease is scarcely attended with any serious danger to life, and it almost always terminates in recovery.

Treatment.—The patient should remain in bed, and in the presence of pain affusions of **lead-water** should be made to the nodules. **Salicylic acid** and **sodium salicylate** have been recommended for the relief of the articular swelling, although personally I have scarcely observed any positive results from their use.

MULTIFORM EXUDATIVE ERYTHEMA.

Symptoms, Diagnosis, and Prognosis.—Multiform erythema frequently sets in with **chill** and **fever**, while at the same time the **spleen** becomes enlarged, and occasionally also the **liver.** **Red spots** then appear upon the **dorsum of the hands and the feet,** and subsequently also upon the forearms and the legs, and, finally, also upon other portions of the body, and occasionally even upon some of the mucous membranes (mouth, pharynx, vagina). The spots are often not larger than a lentil, but increase in size, and are soon transformed into red nodes, over which the skin is tense and glistening. The spots become bluish-red within a short time, so that the hands and feet appear as if covered with frost-bite. Gradually changes in color take place in the nodes similar to those in cases of nodose erythema, so that the nodes acquire a brownish, greenish, bluish, and yellowish color, and finally disappear with slight desquamation.

Should adjacent nodes coalesce into tortuous convolutions, the condition is designated *gyrate erythema.* Occasionally the nodules become pale at the center, while only the periphery remains discolored—*annular erythema.* It may also happen that circles of new nodes form about a node—*erythema iris* or *mamillatum.* Should vesicles appear upon the peripheral nodes, the condition of *herpes iris* will have developed. At times pustules appear upon the nodes, and there may then be danger of confounding the disease with small-pox.

The patients frequently complain of **prickling, burning,** or **sticking** in the nodules. Occasionally **articular swelling and pain,** and in rare instances, in connection with the latter, also **acute endocarditis,** occur. Under such conditions **ankylosis** and **valvular disease of the heart** may result as sequels. The disease terminates, as a rule, within from four to eight weeks in recovery. A fatal result occurs but rarely, but may take place in consequence of complicating pneumonia, meningitis, or nephritis.

Etiology.—Multiform erythema is probably an **infectious disease.** It occurs not rarely in the spring and the autumn in *epidemic distribution,* and exhibits the clinical characters of an infectious disease. The infectious agents are as yet unknown. The disease attacks with preference pallid and delicate *women.* It generally occurs as an independent disorder; less commonly it develops in the sequence of other infectious diseases, as, for instance, syphilis.

Anatomic Alterations.—The anatomic alterations are the same as those in cases of nodose erythema, except that the epidermis is more actively involved, and here accumulation of round cells and swelling and multiplication of epidermic cells are encountered.

Treatment.—The treatment is identical with that of nodose erythema (p. 168).

RECURRING SCARLATINIFORM ERYTHEMA.

Symptoms.—This disorder generally sets in with **fever**, and is attended with **diffuse redness of the skin**, which generally appears earliest upon the chest, then extends over the entire body, and frequently avoids only the face. In the course of a few days desquamation takes place. After an uncertain period of time the manifestations are repeated, and in some persons as many as one hundred recurrences take place in the course of several years. The mucous membranes also may be involved; **difficulty in swallowing** especially is frequently complained of. Occasionally **articular swelling, albuminuria,** and **edema** occur.

Diagnosis.—The disease is suggestive of scarlet fever, although the general condition is but little affected. The disorder may be confounded also with drug-exanthems.

The **treatment** consists in the use of **tepid baths** and inunctions of **carbolated ointment** (1 : 20).

ACRODYNIA.

Acrodynia probably results from the ingestion of *spoiled cereals*, and has been repeatedly observed in *epidemic distribution*. *Gastro-enteritic symptoms* (vomiting, colic, diarrhea) occur first; then *erythematous lesions of the skin* appear, beginning in the hands and gradually extending to the arms and over the entire trunk. At the same time wheals and vesicles form. Subsequently desquamation occurs, but a dark, soot-like *discoloration of the skin* persists, particularly upon the chest and the abdomen. In addition, *nervous symptoms* are present, as, for instance, a crawling sensation, a fur-like sensation, tremor and paralysis of the extremities and the bladder. The duration of the disease, which not rarely has terminated fatally, may be extended over several months.

PELLAGRA.

Pellagra results from the ingestion of spoiled maize, and occurs especially in northern Italy in frightful distribution. The symptoms, as in acrodynia, consist in changes in the skin, gastro-enteritic disturbances, and nervous manifestations. There appears, generally in the months from April to June, upon portions of the body exposed to the air (dorsum of the hands, forearms, chest, back, neck, face), erythematous discoloration of the skin, which, after a short time, undergoes desquamation, and remains of a dark olive-colored hue. The skin becomes fissured and covered with ulcers and crusts. The patients are seized with diarrhea, and complain of a fur-like sensation, a sense of crawling, paralysis, and contractures in the arms and the legs. Ptosis, diplopia, pigmentary retinitis, optic atrophy, and hemeralopia often occur. The patients frequently become psychopathic, are seized with religious mania, and exhibit suicidal tendencies. The disease pursues a chronic course and often terminates fatally within ten or twelve years. In the *skin* dilatation of the blood-vessels, accumulation of round cells about the blood-vessels, the presence of round cells among the epidermic cells, and swelling and proliferation of the latter have been found. In the spinal cord degeneration in the posterior columns and in the lateral pyramidal tracts has been observed. Recovery can be expected only in recent cases. The patient should be provided with a suitable *dwelling* and a *nutritious, mixed diet.* Preparations of *arsenic* and *sulphuric ether* have also been recommended.

VESICULAR INFLAMMATIONS OF THE SKIN (VESICULAR DERMATITIDES).

ECZEMA (MOIST TETTER).

Symptoms, Diagnosis, and Prognosis.—The principal peculiarities of eczema are **weeping** and **itching**, and it may therefore be appropriately designated *itching-weeping tetter*. The disease varies in appearance, and accordingly a distinction is made between papular, vesicular, pustular, impetiginous, squamous, and red eczema. The intimate relation of these various forms is indicated by the fact that frequently the one passes over into the other.

Papular eczema is attended with the presence of innumerable small nodules (papules) upon the affected skin, which occasionally are more easily detected by passing the hand over the surface than upon visual inspection. At the same time the skin is reddened in recent cases, feels hot, and appears puffy and edematous. The patient complains of burning, prickling, and painful itching of the skin.

Vesicular eczema is attended with the formation of vesicles as large as a pin's head, and somewhat larger, which are often crowded together in large number, and contain transparent, serous fluid. If the contents of the vesicles are rich in round cells, the vesicles become opaque and purulent, and the condition is one of *pustular eczema*. Often coalescence of adjacent pustules takes place. Should the contents of the vesicles undergo desiccation, thin honey-yellow crusts form, and the condition is then designated *encrusted eczema*, while the desiccation of pustules gives rise to the formation of thick grayish or greenish-gray crusts, and the condition is therefore designated *impetiginous eczema*. Occasionally the inflamed skin is reddened and moist, and the designation *red* and *weeping eczema* is employed. If the inflamed skin is covered with thin scales, the condition is designated *squamous eczema*.

Eczema may be *generalized* or *circumscribed*. Occasionally in cases of generalized eczema scarcely any portion of the body is uninvolved. Generalized eczema often sets in with chilliness, even with a chill and fever. The patients appear pale and distressed, and also feel profoundly ill. Also, in cases of circumscribed eczema fever and general malaise are not rarely present. *Circumscribed eczema* may appear at various parts of the body.

Eczema of the scalp is frequently dependent upon *head-lice—Pediculi capitis*—and is often of an impetiginous and encrusted character. If the pediculi are themselves not visible, grayish-white nodules should be looked for upon the hairs, as these represent the *ova* (*nits*). In persons of uncleanly habits in whom the hair has been neglected for a long time the hair becomes matted, and constitutes an almost inextricable, offensively smelling meshwork made up of crusts, pediculi, ova, and hairs, which has been designated *plica polonica*. In many places the eczema extends beyond the boundaries of the hair to the adjacent skin of the face, the neck, and the nucha, by which it is distinguished from a seborrhea of the scalp, which always confines itself strictly to the boundaries of the hair. Often adjacent lymphatic glands are enlarged and painful, but only rarely does suppuration, with rupture externally, take place. If encrusted eczema terminates in recovery, it is often at first transformed into squamous eczema. In other instances, however, *squamous eczema of the scalp* develops as an independent and often an exceedingly obstinate disorder. The scalp frequently is profusely covered with grayish-white scales, beneath which the skin is reddened. The scales are abundantly detached in combing the hair, and collect in large amounts upon the clothing. Squamous eczema of the scalp is distinguished from *psoriasis of the scalp* by the fact that in cases of the latter spots of psoriasis are encountered also upon other portions of the skin.

Eczema of the face occurs with especial frequency in infants, particularly at the period of dentition. It often assumes the character of an impetiginous or an encrusted eczema, and the entire face may be covered with crusts, so that the children appear as if concealed behind a mask. The disorder, therefore, is designated also larval porrigo. Occasionally the condition is one of red eczema; the skin of the face is reddened, and appears as if covered with a thin varnish, which is sticky to the touch.

Eczema of the ear occurs especially upon the posterior aspect of the auricle and the lobule of the ear. Not rarely it extends into the external auditory canal, and causes a discharge from the ear. Naturally, an aural discharge may have existed first, and be followed by eczema of the auricle. The condition is frequently one of impetiginous or encrusted eczema, which is attended with marked swelling of the auricle.

Eczema of the nose is frequently situated upon the nasal mucous membrane, is attended with pain and the formation of fissures, readily causes nasal obstruction through swelling of the nasal mucous membrane, and not rarely constitutes the point of origin for recurrent attacks of erysipelas.

Eczema of the lips is attended with thickening of the lips, and often causes severe pain, especially through the formation of fissures.

Eczema of the beard may be readily confounded with sycosis, although the latter disease, which is due to inflammation of the hair-follicles, remains confined to the boundaries of the beard, while eczema often extends beyond these.

Eczema of the nucha is not rarely encountered in fat infants. The condition is frequently situated between the horizontal folds of skin, which readily become gangrenous if cleanliness is not observed. Occasionally eczema of the nucha arises by extension from the head, particularly when this is due to pediculi.

Eczema of the breast occurs about the nipple, particularly in nursing women. Fissures develop readily, and are attended with such severe pain that the women are compelled to cease nursing. In obese women eczema develops not rarely beneath the fold of the breast, and may be the source of a disagreeable rancid odor.

Eczema of the umbilicus likewise occurs especially in the obese.

Eczema of the genitalia occurs in men not rarely upon the scrotum, with involvement of the adjacent surfaces of the thigh, and also upon the dorsum of the penis. The diseased parts become inflamed and swollen, and are not rarely greatly increased in size. In women the greater labia and the mons veneris are involved with especial frequency, and these likewise may be greatly swollen. Occasionally the eczema extends to the mucous membrane of the vagina, and gives rise to leukorrhea. Conversely, however, leukorrhea is occasionally present first, and gives rise to eczema of the genitalia.

Eczema of the anus is an exceedingly troublesome disorder. It frequently gives rise to burning, and, in the presence of fissures, to pain. Occasionally, in consequence of extension of the inflammatory process to the mucous membrane of the rectum, mucopurulent discharges take place from the rectum, and even prolapse of the rectum occurs.

Eczema of the extremities develops at times in the upper, at other times in the lower extremities, and at still other times in both situations simultaneously. Often it appears upon both sides of the body in almost symmetric distribution. *Interdigital eczema* is exceedingly troublesome, and may readily be confounded with scabies, but the acarus and its burrows and ova are not present. Should the disorder extend to the nails, these may be exfoliated. *Eczema of the palm of the hand* may readily give rise to painful fissures, and often compels the patient to keep the fingers flexed. Fissures also develop frequently in the presence of eczema at the flexures of the elbow and of the knee, so that all movement at the affected joints is attended with severe pain. *Eczema of the legs* is frequently red and weeping in character, and is known as *saline discharge*. The inflamed skin is reddened and glistening, and secretes a viscid, sticky fluid.

Eczema is rarely attended with danger to life. Nevertheless, death may result from progressive exhaustion. Chronic eczema is occasionally attended with *albuminuria*, which perhaps is due to the absorption of toxic substances from the inflamed skin and their elimination through the kidneys, in consequence of which the latter are irritated and inflamed. All varieties of eczema have a great tendency to *relapse*, and often are extremely resistant to treatment. In accordance with the *duration of the disease* a distinction can be made between acute and chronic eczema. Recovery from acute eczema may take place within from two to four weeks, while chronic eczema may persist for months, years, and occasionally throughout the whole of life.

Anatomic Alterations.—In the presence of eczema the blood-vessels of the skin are found dilated, and in places surrounded by accumulations of round cells. The round cells wander in part among the cells of the epidermis, which themselves become swollen, and the skin is raised in places in the form of small nodules or papules. Vesicular eczema develops if the alterations described are attended with profuse escape of blood-plasma from the vessels of the cutis, causing elevation of the horny layer from the mucous layer. The vesicles possess an alveolar structure, the individual septa consisting of compressed epidermic cells. If large numbers of round cells are admixed with the serum of the vesicles, partly by immigration from the vessels of the cutis, and partly from multiplication of the epithelial cells of the mucous layer, a pustule results, and accordingly the condition becomes one of pustular eczema. In cases of chronic eczema the cutis is sclerotic, traversed by numerous collections of round cells, and its blood-vessels and lymphatics are dilated. The sebaceous and the sudoriferous glands are partially destroyed. The cellular infiltration not rarely extends into the subcutaneous connective tissue.

Etiology.—Eczema is one of the *commonest diseases of the skin.* Its causative factors are either external (local) or internal. Among the external causes are mechanical, chemic, and thermic irritants. Among the **mechanical injurious influences** are persistent chafing and rubbing of the skin in one situation. For this reason itching cutaneous exanthems not rarely give rise to eczema, especially prurigo, pruritus, pediculosis, scabies, etc. Some individuals suffer from eczema on putting on new and stiff underclothing. Occasionally eczema develops between the buttocks in novices in riding, and after long marches—so called eczema intertrigo. The wearing of a straw hat also may give rise to eczema of the forehead. **Toxic eczema** may result from the application to the skin of balsamics, ointments, spirituous lotions, from contact of the skin with alkalies, acids, and other irritating substances. Under certain conditions eczema occurs as an **occupation-disease**, as, for instance in masons, bakers, laundresses, and also in physicians from the employment of carbolic acid and mercuric chlorid. Occasionally irritating discharges from the ear, the nose, the genitalia, or the anus, excite eczema upon adjacent parts of the skin. **Caloric eczema** occurs not rarely in those engaged in agricultural pursuits if the uncovered neck and arms are exposed for a long time to the rays of the sun. The significance of **internal causes** in the development of eczema is known only as a result of clinical experience. Obstinate eczema develops not rarely in cases of *nephritis* and *diabetes mellitus*, perhaps in consequence of autointoxication. Chronic *disease of the stomach and the intestines* and *gout* also may be followed by eczema. *Scrofulous* and *rachitic* individuals likewise suffer frequently from eczema. It is believed that eczema may result through reflex influences in consequence of *disease of the female generative apparatus.* The influence of the nervous system is thought to be indicated by the fact that some persons are attacked by eczema after *fright* and *emotional disturbances.* Pallid, delicate, and obese persons exhibit a greater tendency to eczema than others.

Treatment.—The treatment must, in the first place, be causal. **Symptomatic treatment** will vary in accordance with the variety of eczema. Papular eczema should be treated with cool affusions of lead-water, or solution of lead subacetate, followed by inunction of the skin with a pure oil, as, for instance, olive-oil. Vesicular and pustular eczema will only require treatment with indifferent fats. In cases of impetiginous and encrusted eczema the accumulations upon the skin should first be removed by generous application every two hours of olive-oil or cod-liver oil, and then a thin layer of the following ointment upon linen should be

℞ Simple plaster,
Simple litharge-plaster, each, 25.0 (¾ ounce);
Olive-oil sufficient to make a soft plaster.
Apply externally.

The linen and the ointment should be renewed once daily. The former should be soft, and preferably old, though clean. In cases of squamous eczema the ointment just named may likewise be employed. In obstinate cases the skin should be painted daily with tar, as, for instance:

℞ Oil of birch,
Olive-oil, each, 25.0 (¾ fluidounce).—M.
To be applied daily.

At places where inflamed surfaces of skin come in contact and rub against each other, lint that has been smeared with the ointment named should be interposed.

MILIARIA.

Symptoms, Diagnosis, and Prognosis.—Miliaria is attended with the formation of small vesicles up to the size of a pin's head, containing at times clear, watery fluid and resembling a dew-drop—*crystalline miliaria;* at other times surrounded by a reddened areola—*red miliaria;* and at still other times, finally, containing fluid rendered turbid from admixture with colorless blood-corpuscle—*white miliaria.* The contents of the vesicles are either neutral or alkaline in reaction. The vesicles are often packed together in innumerable amount, and occur especially upon the covered portions of the body, as, for instance, the breast and the abdomen. On passing the fingers over the skin they can be readily felt as small elevations. Within a few hours or days the vesicles undergo desiccation, and slight desquamation of the skin takes place. The exanthem causes little inconvenience, and many patients are unconscious of its existence.

Etiology and Anatomic Alterations.—Miliary vesicles occur after **profuse sweating,** the perspiration either being collected beneath the skin at the excretory duct of the sudoriferous gland (crystalline miliaria), or between the horny layer and the mucous layer of the epidermis (white and red miliaria). After active physical exertion, after courses of sweating, the application of warm affusions, in cases of pulmonary tuberculosis, during the crisis of acute infectious diseases, in the stage of defervescence of typhoid fever, in cases of miliary fever, and in conditions of collapse (Asiatic cholera, death-agony) profuse sweating and the formation of miliaria may readily take place.

Treatment.—No treatment directed especially against the exanthem is necessary. The indication particularly is to prevent profuse sweating, and this may be accomplished most certainly by means of atropin and cold ablutions.

CHEIROPOMPHOLYX.

Cheiropompholyx generally occurs only during the *summer*, and is the result of excessive *sweating*. The disease appears principally on the *palms of the hands*, less commonly also on the soles of the feet. In these situations there form beneath the horny layer of the epidermis *vesicles* varying in size up to and beyond a pin's head, which on puncture discharge clear fluid of alkaline reaction and suggestive of the appearance of sago-granules. The disorder is frequently recurrent, but is unattended with danger. The vesicles should be opened with a knife, and a powder such as the following be applied:

℞ Salicylic acid,
Zinc oxid,
Talcum, of each, 10.0 (2½ drams).—M.
Use as a dusting-powder.

BULLOUS INFLAMMATIONS OF THE SKIN (BULLOUS DERMATITIDES).

PEMPHIGUS.

Symptoms, Diagnosis, and Prognosis.—Pemphigus is attended with the formation of blisters upon the external integument, which at first contain clear fluid of neutral reaction, subsequently becoming turbid and alkaline. The vesicles vary in size between that of a pea and that of the palm of the hand. Recovery takes place either by desiccation of the contents of the vesicle generally to a thin crust, which after a time falls off and leaves behind a cicatrix, or the blisters rupture and the reddened and weeping corium is exposed, and this gradually becomes covered with new epidermis. Brownish areas then remain upon the skin, which, however, may entirely disappear in the course of years. Occasionally vesicles of pemphigus appear upon the *mucous membranes*, as, for instance, that of the cheeks, the pharynx, the larynx, the bronchi, and even upon the conjunctiva. In accordance with the *duration of the disease* a distinction is made between *acute* and *chronic pemphigus*. In cases of acute pemphigus a distinction must be made between that in the newborn and that in adults. Chronic pemphigus occurs occasionally in the form of foliaceous life, and scarcely affects the general condition of the child in any manner, although in other instances fever occurs, and death may result, particularly amid the symptoms of gastro-intestinal catarrh or of pneumonia.

Acute pemphigus of adults often sets in with *prodromes* (chilliness, fever, gastric disturbance). In the course of from one to three days the characteristic vesicles of pemphigus appear, generally associated with prickling, burning, and pain in the skin. Between the vesicles roseolæ and wheals are often present, upon which likewise vesicles subsequently appear. The fever generally rises with the formation of the vesicles, the patients feel miserable and exhausted, and often exhibit enlargement of the spleen and albuminuria. Vesicles upon the buccal mucous membrane give rise to pain and a disagreeable odor from the mouth; those upon the pharyngeal mucous membrane cause difficulty in swallowing; while the formation of vesicles upon the bronchial mucous membrane is occasionally attended with the expectoration of membrane.

Chronic pemphigus not rarely persists for several years, during which vesicles are present only intermittently or continuously. The patient gradually loses strength, amyloid degeneration takes place in various organs, and finally death results from exhaustion.

Foliaceous pemphigus is generally characterized by the flaccidity of the vesicles and the wrinkling of their cutaneous covering. When the blisters rupture the corium is exposed, as a new covering does not form. The patient therefore presents an excoriated appearance, and death results almost unexceptionally, at times more rapidly, and at other times more slowly, in consequence of exhaustion.

Vegetating pemphigus likewise generally terminates fatally, but fortunately is an exceedingly rare cutaneous disorder. It can be recognized from the fact that beneath the covering of the vesicles granulations are encountered, whose appearance suggests that of broad condylomata, and which therefore are frequently considered syphilitic.

Anatomic Alterations.—The vesicles of pemphigus are situated between the mucous and the horny layer of the epidermis. The smaller vesicles present a meshwork constituted of compressed epidermic cells, which is wanting in the larger vesicles. The vesicles contain round cells, red blood-corpuscles, and granular detritus, as well as large numbers of eosinophile cells. The fluid is often of gelatinous consistency. Accumulations of round cells are present in the cutis around the blood-vessels, the hair-follicles, and the sudoriferous glands. Specific bacteria have often been looked for in the contents of the vesicles, but hitherto without success.

Etiology.—Among the causes of pemphigus a distinction can be made between infectious, toxic, autotoxic, and nervous influences. Not rarely pemphigus occurs in the manner of an independent (primary) **infectious disease**; or in other instances it develops as a secondary disorder in connection with some other antecedent infectious disease, such as septicopyemia, ulcerative endocarditis, scarlet fever, measles, varicella, variola, malaria, leprosy, and syphilis. Syphilitic pemphigus occurs particularly in connection with hereditary syphilis in children, and involves especially the soles of the feet and the palms of the hands, and occasionally only these parts. **Autotoxic pemphigus** includes those cases that develop in association with urethritis and derangement in the function of the kidneys. Possibly the *pemphigus of pregnancy* and of *lactation* belongs in this category. **Toxic pemphigus** occurs in some individuals after the ingestion of certain drugs, as, for instance, potassium iodid, salicylic acid, antipyryin. **Nervous pemphigus** is sometimes observed in cases of epilepsy, mental aberration, hysteria, and paralysis.

Treatment.—Causal treatment should be instituted in cases of syphilis, malaria, and acute pemphigus of the newborn. In the last-named event rigid isolation of the patient should be observed. Nothing can be accomplished with internal remedies in the treatment of pemphigus. Daily **baths** at a temperature of 35° C. (28° R. —95° F.), with addition of sodium bicarbonate (from 300 to 500) or of mercuric chlorid (from 5.0 to 10.0), should be prescribed, the vesicles should be punctured by means of a knife, and the skin in these situations covered with a borated ointment (1.0 : 30). In cases of foliaceous pemphigus the patient should remain continuously in a warm bath, and be given a nutritious diet. Injections of **strychnin** (0.005—$\frac{1}{15}$ grain—daily) and intravenous infusion of physiologic saline solution have also been recommended.

HEREDITARY BULBOUS EPIDERMOLYSIS.

This disease is *rare*, and in some families *hereditary*. It occurs in early childhood and persists throughout the whole of life. Its peculiarity consists in the fact that the skin, and with especial readiness during the summer, becomes the seat of vesicles in all places where it is exposed to mechanical irritation through chafing, rubbing, pressure, or the like. Vesicles may appear even upon the mucous membrane of the mouth, as, for instance, after mastication. In all probability the disorder depends upon an hereditary undue readiness of detachment of the horny cells from the prickle-cells of the epidermis. Treatment is ineffective.

EXFOLIATIVE DERMATITIS.

It is wise to distinguish two varieties of exfoliative dermatitis, accordingly as the disorder occurs in the newborn or in adults.

Exfoliative dermatitis of the newborn occurs most frequently in the *second week of life*, and is attended in the mildest cases with marked redness and desquamation of the skin, without disturbance of the general condition, while in the severe cases the epidermis is exfoliated in large sheets, after having been detached in places in the form of flaccid vesicles, and the children appear as if scalded and excoriated. The disorder might be confounded with foliaceous pemphigus if this disorder were not extremely rare in children. Painful fissures readily form on the lip and the nose. The *duration of the disease* generally extends over one or two weeks. Half of the patients die as a result of progressive exhaustion, pneumonia, or diarrhea. The treatment consists in *tepid baths* twice daily and the use of dusting-powders.

Exfoliative dermatitis of adults is attended with redness and often also with weeping of the skin and desquamation of the epidermis. The patients are feverish, gradually lose strength, and die in the course of a few months from pneumonia, diarrhea, or albuminuria. Occasionally paralysis also develops. In a patient under my care the cutis contained a large number of round cells and the epidermis large collections of cocci. The treatment consists in the use of baths and oily inunctions.

PUSTULAR INFLAMMATIONS OF THE SKIN (PUSTULAR DERMATITIDES).

IMPETIGO (ECTHYMA).

Impetigo and ecthyma are the designations applied to all *pustules* upon the skin. When the pustules undergo desiccation greenish and grayish-green scales and crusts remain behind upon the skin. Among the causes for impetigo and ecthyma, mechanical, caloric, chemic, and infectious injurious influences may be mentioned. *Mechanical impetigo* and *ecthyma* develop, for instance, from scratching of the skin, and occur frequently therefore in the presence of itching cutaneous eruptions (prurigo, pruritus, pediculosis, and scabies). *Caloric impetigo* occurs as a result of the action of excessive heat upon the skin. *Toxic impetigo* is observed after inunction of irritant substances (croton-oil, antimony, and potassium tartrate), and in certain occupations, as, for instance, on the forearm in masons and smiths, in consequence of splashing with lime or incandescent particles of iron. *Infectious impetigo* and *ecthyma* constitute the exanthem of variola. They develop also in connection with septicopyemia and ulcerative endocarditis, and are attended at times with febrile symptoms, in the manner of an *independent infectious disease*. *Contagious impetigo* and *herpetiform impetigo* also are to be considered as independent diseases.

Contagious impetigo occurs particularly in children, not rarely in epidemic distribution, and has been observed repeatedly after vaccination with glycerinated lymph. It has been possible to demonstrate infection through personal contact. Often the appearance of the exanthem is preceded for one or two days by *febrile prodromes.* The first pustules generally appear upon the face; then they develop also upon the scalp, the nucha, the trunk, and the extremities. The mucous membranes are but rarely involved. The pustules dry up into crusts, which subsequently fall off without leaving cicatrices. Specific bacteria have not as yet been found in the contents of the pustules. Recovery from the disease generally takes place within from two to four weeks, although relapses occur. In the treatment, *baths* and *oily inunctions* of the skin will suffice.

Herpetiform impetigo is a rare disease of the skin which occurs only exceptionally in men, but with relative frequency in *pregnant* and *recently delivered women.* After a *chill* and *fever pustules* appear upon the *inner aspect of the thighs,* then upon the abdomen, and only much later upon the skin of the face and the arms. The vesicles are arranged in groups and often in circles. Around the individual circles of vesicles new circles form, and each new eruption is generally attended with chilliness and febrile movement. The vesicles dry up into crusts, after the removal of which the skin appears red and weeping, but without loss of structure. Occasionally it exhibits a smeary deposit, at times also granulations (vegetating herpes). In some cases vesicles appear also upon some of the mucous membranes (mouth, larynx, rectum, vagina). The disease generally terminates fatally with exhaustion or septicopyemia. The *treatment* consists in a *nutritious diet, baths,* and *oily inunctions.*

ACNE VULGARIS.

Symptoms, Etiology, and Diagnosis.—It is customary to distinguish several varieties of acne, which are designated disseminated, frontal, cachectic, syphilitic, and toxic, respectively. All, however, agree in the circumstance that they depend upon inflammation of the sebaceous follicles and the hair-follicles.

Disseminated acne is so common a disorder that scarcely anyone wholly escapes. Generally it appears at the period of *puberty,* then frequently disappears at about the twenty-fourth year, although in some individuals it persists throughout life. *Chlorotic* and *scrofulous persons* are attacked with especial frequency. The ingestion of fatty, spiced, and irritating *articles of food* is also believed to favor the occurrence of the disease. Some women are attacked by acne at the *menstrual period* and during *pregnancy* or the *puerperium.* Acne often begins with the formation of a *comedo,* which can be readily recognized as a black dot in the orifice of a sebaceous follicle, and represents only the occluded orifice of such a follicle. If the follicle becomes inflamed, a red nodule forms, with a black dot at its center—*punctate acne.* The nodule becomes gradually transformed into a pustule—*pustular acne;* and if the surrounding tissues are reddened and become hard, the condition is designated *indurated acne.* The nodule may be the seat of severe pain, or even give rise to fever and swelling of adjacent lymphatic glands. At times acne-nodules and acne-pustules are present in such large number as to suggest the appearance of an ear of barley, and the disorder is then designated also *hordeolar acne.* Disseminated acne generally develops with especial profusion upon the forehead, the chest, the back, and the inner aspect of the thighs. The number of efflorescences is susceptible of the widest variation. The disorder is unpleasant on account of the disfigurement of the face, but it is unattended with danger.

Frontal acne is known also as *varioliform acne* or *necrotic acne,* and occurs only upon the forehead, particularly at the junction of the latter with the hair. It is not preceded by the formation of comedones. There develop flat papules, then pustules, covered at the center with a crust, which appears slightly depressed and consequently is suggestive of a variolous pustule. When the crust falls off, a cicatrix often remains.

The designation *urticate acne* has been applied to cases characterized by great itching, which disturbs sleep and causes loss of strength. The disorder may be attended with the presence of pustules upon the trunk and the extremities.

Cachectic acne develops in the sequence of severe diseases (pulmonary tuberculosis, scrofulosis, variola), and depends probably upon hyperplasia of the glandular cells and resulting occlusion of the sebaceous follicles.

Syphilitic acne is the result of syphilis.

Toxic acne develops as a result of the external application or the internal administration of certain medicaments, among which especially the *bromids, iodids, preparations of tar,* and *chrysarobin* may be mentioned. Iodin and bromin have been demonstrated chemically in the contents of the pustules of bromid-acne and iodid-acne. Further, it appears that different individuals exhibit a varying predisposition to the development of acne.

Anatomic Alterations.—In cases of acne the sebaceous follicles and not rarely also the hair-follicles are found filled with pus-corpuscles and granular detritus, while the excretory duct of the follicle is occluded. Not rarely the adjacent cutis also has become involved in the inflammatory process and is infiltrated with round cells. In the majority of cases bacteria are responsible for the inflammation.

Prognosis.—The prognosis of acne vulgaris is favorable, although the disorder often is extremely obstinate and a source of much annoyance on account of the disfigurement to which it gives rise.

Treatment.—**Causal therapy** is indicated especially in cases of toxic and cachectic acne. **Symptomatically,** comedones should be removed, and the simplest means is by compression. In addition, the skin should be washed with **green soap,** which causes active desquamation. Acne-pustules should be opened with a bistoury and should then be washed with a **solution of mercuric chlorid** (0.1 : 100). If the skin surrounding the pustules is infiltrated, **warm cataplasms** should be applied, and on the appearance of fluctuation the collection of pus should be opened and its cavity cleansed with a solution of mercuric chlorid. For the relief of bromid-acne, washing of the skin with **camphor-water** and administration of **solution of potassium arsenite** (5 drops thrice daily) have been recommended.

The number of remedies recommended for the treatment of acne vulgaris is extremely large. Covering the skin with *mercurial plaster* or the *diachylon plaster* of Hebra, and the employment of sulphurous pastes and lotions, may further be mentioned.

ACNE MENTAGRA.

Symptoms and Diagnosis.—Acne mentagra, also designated sycosis, results from suppurative inflammation of the hair-follicles of the beard, less commonly of the hair of the head, the axillary cavity, the mons veneris, the nasal entrance (vibrissæ), or the eyebrows. The hairs in the diseased follicles are embedded in a small collection of pus, can generally be extracted easily and without pain by means of forceps, and their follicular portion is swollen and softened. Often the adherent hair-sheath is infiltrated with pus-cells. If potassium hydroxid is added to a microscopic preparation, and a short time is permitted to elapse, until the specimen has become transparent, round spores and long, fungous filaments of the Trichophyton tonsurans will sometimes be observed to come into view (Fig. 31). Naturally, it is often necessary to examine numerous hairs before one exhibiting the fungus is found. There is, therefore, a variety of acne mentagra that is nothing more than a manifestation of herpes tonsurans of the hair of the beard, for herpes tonsurans likewise is the result of invasion by the Trichophyton tonsurans. Nevertheless, trichophyton sycosis is rather one of the less common varieties, the majority of cases probably occurring as a result of the action of pyogenic bacteria. Adjacent pustules readily coalesce, and, in consequence of desiccation, form crusts, from which the hair projects. If the crusts are removed, nodules and infiltrations of the skin often come into view, which are suggestive of the appearance of condylomata and are traversed by numerous collections of pus. In contradistinction from eczema of the beard, the changes are always confined to and do not extend beyond the boundaries of the hair. The patients complain frequently of pain, and are especially annoyed by the disfigurement. If the follicles are profoundly affected, areas of alopecia may appear in the beard after recovery. The disease is often most obstinate, and recurrence readily takes place.

Etiology.—Trichophyton sycosis results from infection by a domestic animal (horse, dog, cow) or from a diseased human being. In the latter event inoculation not rarely takes place through the intermediation of a barber-shop in which the razors, combs, brushes, or scissors have been used upon a case of trichophyton sycosis without being thoroughly disinfected subsequently. Occasionally physicians are inoculated in examining patients suffering from sycosis. Little is known with regard to the causes of other varieties of sycosis. The use of a dull razor, excessively dense growth of the hair, antecedent eczema, chronic coryza, and, in cases of coryza, the irritation from snuff in those who use it have been considered among the etiologic factors. The disorder occurs almost exclusively in men with beards.

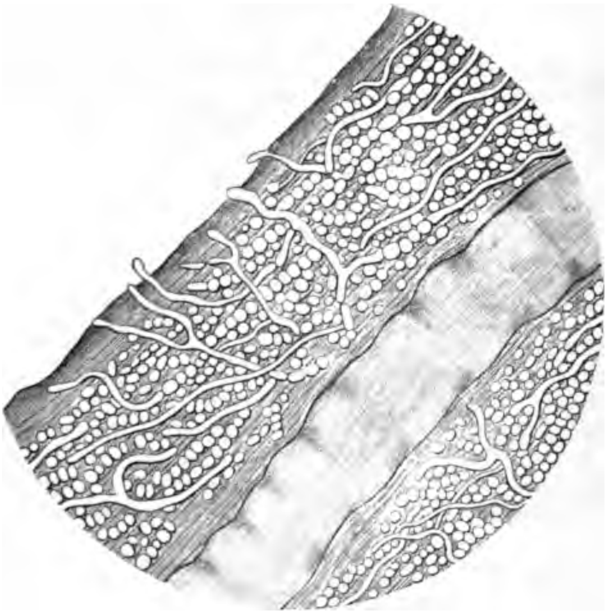


FIG. 31.—Hair from a nodule of parasitic sycosis, at one point presenting especially fungous spores; treated with potassium hydroxid; magnified 275 times (personal observation, Zurich clinic).

Prognosis.—The prognosis of acne mentagra is good, as there is no danger to life, and with sufficient patience the disorder can almost always be cured.

Treatment.—In a case of *trichophyton sycosis* **olive-oil** should be applied every two hours to such crusts and scales as may be present, and these should additionally be covered with a bit of flannel saturated with oil until the crusts fall off. The *beard* should, of course, have been previously *cut short,* in order that the oil may penetrate the crusts. After removal of the crusts the affected parts should be painted twice daily with **oil of turpentine**, in order to destroy the fungi. In cases of ordinary sycosis also it is necessary to cut the beard short and to remove the crusts by means of olive-oil. Such pustules as may be present are opened with a knife, and the inflamed surfaces are washed twice daily with a solution of mercuric chlorid (0.1 : 100). In obstinate cases it will be necessary to remove by means of forceps all hairs arising from pustules. This procedure must often be continued for many days. In addition, the beard must be shaved regularly. Such infiltrates and nodules as may be present should be covered with **mercurial plaster**.

The designation *frambesiform sycosis* has been applied to infiltrates and nodules that occur at the junction of the nucha and the hair, are penetrated by hairs, and probably result from irritation of the skin by stiff collars. It has not rarely been necessary to cauterize, to scrape, or to excise the nodules.

Etiology.—The disorder is often dependent upon excessive **indulgence in alcohol**, and those who drink whisky suffer more commonly than those who drink wine, and the latter in turn more commonly than those who drink beer. The disorder develops not rarely in the sequence of chronic **disease of the stomach, the intestine, or the liver**, and under such circumstances the possibility of auto-intoxication has been considered. In women acne rosacea may develop in the presence of **disease of the generative organs**, particularly of the uterus, but also during pregnancy or following parturition. Under such circumstances reflex influences are possibly operative. Some **occupations** favor the occurrence of acne rosacea in consequence of exposure of the skin repeatedly and constantly to the external air, or to radiating heat (smiths, locksmiths, masons, farmers, hunters, soldiers). Not rarely several of the injurious influences named may be operative at the same time. Men are attacked generally after the thirtieth year of life; women frequently as early as puberty. The disorder occurs but rarely in childhood.

Diagnosis.—The disease can be readily recognized, and is distinguished from **lupus** and **carcinoma** by the absence of ulcerative destruction, from **syphilis** by the absence of other syphilitic manifestations, and from **freezing** by its development independently of the action of cold.

Prognosis.—Acne rosacea is not a dangerous disease, but it is disfiguring, and exposes some persons unjustly to the suspicion of alcoholism.

Treatment.—The treatment must be both **causal** and **local.** The former is directed against the etiologic factors in the individual case, but is surrounded with difficulty in drunkards because of their unwillingness to abstain from alcohol. In mild cases of acne rosacea affusions of *lead-water* should be made for two hours morning and night. In advanced cases a bit of leather upon which a layer of *mercurial plaster* has been spread should be applied at night; *puncture of the skin*, *electrolysis*, and *inunctions of oil of turpentine* have also been recommended. If the disease be marked, the disfiguring nodules, lobules, or lobes should be removed with the *knife.*

SCALY INFLAMMATIONS OF THE SKIN (SQUAMOUS DERMATITIDES).

PSORIASIS.

Symptoms and Diagnosis.—Psoriasis, next to eczema, is *one of the most frequent of the diseases of the skin.* It is attended with the collection of **grayish** or **glistening-white scales** upon the skin, which are suggestive of the appearance of asbestos or mother of pearl, and are situated upon a **reddened base.** Often the redness of the skin extends beyond the collection of scales, so that the latter is surrounded by an areola several millimeters in extent. The scales can be readily detached and removed with the fingernail, permitting the reddened skin to come into view with especial distinctness, and this exhibits a marked tendency to hemorrhage. The earliest changes in the skin generally take place at such points as are exposed to pressure and friction, especially the elbows and the knees. The external and the internal aspect of the auricle and the scalp also are often involved early. If the hair of the head is separated, an extensive layer of thick, superimposed, glistening-gray scales comes into view, beneath which the scalp is dry and reddened. The patient will have noticed for some time that in combing his hair large numbers of scales remain upon the comb. Gradually accumulations of scales form upon the trunk, the face, and other parts of the body. The palms of the hands and the soles of the feet generally remain uninvolved, although, on the contrary, they are not rarely the seat of syphilitic psoriasis. The nails also may be affected; white spots appear upon them, and they become fibrillated and friable. The accumulations of scales are frequently most numerous and most dense at places where the trunk is exposed to mechanical irritation, in women often at the junction of the chest and the abdomen, in consequence of constriction by the clothing. The *appearance and the further development of the plaques of psoriasis* can frequently be observed in the same patient. The most recent alterations often consist in punctate accumulations of scales, so that the designation *punctate psoriasis* has been employed. In consequence of gradual growth *guttate psoriasis* and *nummular psoriasis* result, accordingly as the mass of scales attains the size of a drop or of a coin. Often adjacent collections of scales coalesce and may follow a convoluted, tortuous arrangement—*gyrate psoriasis* (Fig. 32). Not rarely the coalescence takes place in such a manner that a closed circle of plaques of psoriasis results, the middle of which is constituted by unaltered skin—*orbicular psoriasis.* If the skin is covered by a large coherent surface of scaly masses, the condition is designated *diffuse psoriasis,* and if (in cases of long standing) the scales acquire a blackish color, the designation *psoriasis nigra* or *nigricans* is employed.

Psoriasis frequently develops without any other morbid disturbances. In other instances slight febrile movement and pallor occur. At times pain and swelling, and subsequently deformity, also appear in some joints. Some patients complain only of the disfigurement. Others are distressed by the itching, burning, and

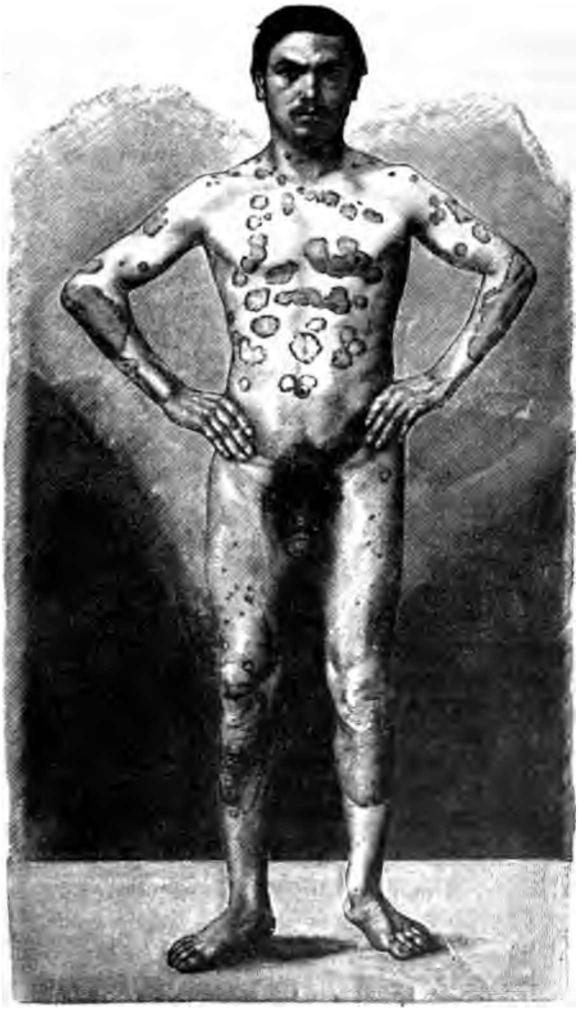


Fig. 32.—Extensive psoriasis in a man, 28 years old; from a photograph (personal observation, Zurich clinic).

pain. In cases of diffuse psoriasis the movements of the joints are not rarely interfered with, and painful fissures may readily form. Psoriasis generally pursues a *chronic course*, and, untreated, persists for several years. Although it is curable, *recurrence* takes place as a rule. Nephritis and albuminuria may appear after long-continued psoriasis.

Serious diagnostic difficulty arises but seldom. In cases of **syphilitic psoriasis** the scales are less abundant, the plaques appear especially upon the palms of the hands and the soles of the feet, and other syphilitic lesions are present besides. In contradistinction from **eczema**, weeping and marked itching of the skin are wanting with psoriasis. **Pityriasis rubra** is characterized by its unfavorable course and the complicating marasmus. **Lichen ruber** is attended with the presence of small efflorescences, generally as large as the head of a pin, arranged in groups side by side, and followed by slight desquamation.

Anatomic Alterations.—Psoriasis is attended with elongation and expansion of the papillæ of the cutis, and dilatation of the blood-vessels, which are surrounded by accumulations of round cells. The lowermost layer of the cells of the mucous lamina of the epidermis, arranged in the form of palisades, is in a state of proliferation, while the remaining cells have lost their thorn-like processes.

Etiology.—Psoriasis is frequently an **hereditary disease**, although the disorder but rarely appears as early as childhood. A **toxic variety of psoriasis** is also believed to exist, resulting from the employment of *borax*. Isolated observations of **inoculation** are on record, although nothing is as yet known with regard to a germ of psoriasis. In many cases it is impossible to elicit a cause for the disorder, which has been attributed to unsuitable food, excessive indulgence in alcohol, and the like.

Prognosis.—Psoriasis is, as a rule, unattended with danger to life, although it is often an obstinate disorder, concerning which one can never know whether it will remain permanently cured.

Treatment.—In the treatment of psoriasis, **internal and external remedies** should be combined. Among internal remedies, arsenic is deserving of the warmest commendation:

℞ Bitter-almond water,
Solution of potassium arsenite, each, 5.0 (75 minims).—M.
Dose: 10 drops thrice daily after eating.

In addition, the patient should take a sulphur-bath daily, and subsequently remove the scales upon the skin by means of a stiff brush that has been dipped in a soap of sulphur and tar. The *sulphur-bath* should have a temperature of 35° C. (28° R.—95° F.), and contain 150 grams of potassium sulphid. The patient should remain in such a bath for from half an hour to an hour, but he must be watched by an attendant, in order that he may not, unobserved, be overcome by the vapors of hydrogen sulphid and be drowned. The following formula may be employed as a sulphur-tar soap:

Green soap,	
Liquid pitch,	
Precipitated sulphur,	
Dilute alcohol,	each, 25.0 (⅞ ounce).—M.

Apply externally.

Should pain and excessive redness result from active irritation of the skin, the tar-soap should be omitted for a few days and be replaced by inunctions of borated ointment:

℞ Boric acid,	3.0 (45 grains);
Wool-fat,	
Lard,	each, 25.0 (⅞ ounce).—M.

For external application.

For the treatment of so annoying and so frequent a disorder as psoriasis a large number of remedies have naturally been recommended, of which a few may yet be mentioned. Internally, large doses of *potassium iodid*, *thyroid extract*, and *salicylic acid* have been recommended, although I have observed no good results from any of these drugs. Of external remedies, *pyrogallic acid* and *chrysarobin* especially may be mentioned, both employed in the form of ointments containing from 5.0 to 10.0 (75 to 150 grains) to 50.0 (1½ ounces). Courses of treatment at sulphurous or indifferent *baths* have been much prescribed.

PITYRIASIS RUBRA.

Pityriasis rubra is an uncommon disease of the skin, concerning whose etiology nothing is known. The disorder occurs most frequently in men. The affected skin becomes reddened, and undergoes desquamation. The latter is generally branny, and occurs but rarely in the form of large scales. At times the skin becomes thin and, to a certain degree, unduly tense, so that ectropion of the eyelids develops, and the movements of the hands and the fingers are interfered with, the latter assuming a persistent semi-flexed position. The skin exhibits a tendency to fissures; at times the hair falls out. The disease either involves the entire body simultaneously, or arises from several points upon the skin, and gradually extends over the entire body. The patients at the same time gradually fail in strength, are not rarely attacked by tuberculosis of the lungs or tuberculosis of the lymphatic glands, and death frequently results from marasmus. In the diseased areas of skin dilatation of the vessels of the cutis, accumulation of round-cells, and flattening and atrophy of the papillæ have been found. The epidermis, and the sebaceous and sudoriferous glands also have been atrophied. Occasionally, many pigment-cells have been present in the cutis.

The **treatment** consists in **tepid baths** and **oily inunctions**, while internally **potassium iodid** may be employed.

PAPULAR INFLAMMATIONS OF THE SKIN (PAPULAR DERMATITIDES).

PRURIGO.

Symptoms, Diagnosis, and Prognosis.—Prurigo is attended with the formation of **nodules (papules)** upon the skin, which are associated with excessive **itching (pruritus)**. As a rule, the disorder begins in earliest childhood, most frequently toward the *close of the first year of life*, and appears earliest on the legs, subsequently extending to the thighs, the forearms, the upper arms, and even the trunk and the face. Papules are generally wanting upon the scalp, but here active formation of scales takes place. The flexor aspects of the joints always escape. In the incipiency of the disease repeated attacks of *urticaria* occur. Then small nodules appear beneath the epidermis, which at first can be better felt by passing the fingers over the skin than they can be appreciated with the eye. Gradually these papules project distinctly above the surface of the epidermis, and the skin often feels rough like a grater. The *papules* are in part pale, and in part red, and when punctured and compressed discharge a clear fluid. They are often covered by epidermic scales. The patient is, at the same time, harassed by intolerable *itching*, which is especially aggravated by heat, and therefore becomes particularly distressing at night in bed, preventing sleep, and resulting in emaciation and exhaustion. Some clinicians contend that the itching precedes the formation of the papules. The persistent and vigorous scratching leads to the formation of crusts, and even of pustules upon the skin. After the disease has existed for some time the skin acquires a brownish discoloration (melasma), in consequence of free bleeding from the scratching, and a chronic inflammatory, infiltrated, and thickened character. Frequently adjacent lymphatic glands become enlarged—*prurigo buboes;* the inguinal lymphatic glands especially not rarely appear as enormous packets beneath the skin. The patients slowly undergo progressive emaciation. Occasionally **albuminuria** develops. The disease generally pursues a *chronic course*, and not rarely persists throughout life. Recovery can be expected only if the disorder has not existed for more than four years. Often numerous remissions and exacerbations occur, and improvement is especially prone to take place during the summer.

Anatomic Alterations.—Little of a definite nature is known with regard to the anatomic alterations of prurigo. Circumscribed swelling and enlargement of the papillary bodies of the cutis, together with cellular proliferation, and also multiplication of the cells in the Malpighian layer, have been described.

Etiology.—Prurigo is not rarely an *hereditary disease.* In addition, **unsuitable food, rachitis, scrofulosis, pulmonary tuberculosis,** and **pseudoleukemia** have been mentioned as causative factors.

Treatment.—Innunctions of the skin with **green soap** should be made at night, and a **sulphur-bath** (100.0 potassium sulphid to a full bath at 35° C.—95° F.—of two hours' duration) should be taken in the morning. After the bath the skin should be anointed with carbolated ointment (5 : 50).

In addition, subcutaneous injections of *pilocarpin* and the treatment applicable to psoriasis have been recommended.

LICHEN OF THE SCROFULOUS.

Symptoms and Diagnosis.—Lichen of the scrofulous is attended with the presence of papules about the size of a pin's head, from pale red to brownish-red in color, with a small thin crust at the center. If the latter be removed, a hair will be found at the center of the papule, and whose follicle exhibits a clearly defined, slightly elevated boundary. The papules are unassociated with other symptoms. The first papules generally appear upon the back, particularly in the scapular region, upon the chest, and in the hypogastrium. Subsequently the extremities, the trunk, and the head also are involved. Upon the arms the flexor surfaces especially are affected. It is distinctive for the papules to be arranged in groups or in circles. The disorder occasionally persists for years.

Anatomic Alterations.—Microscopic examination of the skin discloses round-cell accumulations in the vicinity of the hair-follicles, plugging of the latter with round cells, and obstruction of the excretory ducts with accumulated epidermic cells.

Etiology.—The disease generally occurs in **scrofulous children,** less commonly in those with pulmonary tuberculosis, and but rarely after the twentieth year of life. The patients generally present a pale and fatty skin.

Prognosis.—The disorder is unattended with danger, and is curable.

Treatment.—The skin should be anointed several times daily with **codliver-oil** and also internally 25 c.c. of the same oil should be administered daily.

LICHEN RUBER.

Symptoms, Diagnosis, and Prognosis.—Lichen ruber is attended with the formation upon the skin of red papules averaging in size that of a pin's head, which are at times acuminated and covered with a thin scale of skin—*acuminated lichen ruber;* and at other times are rather flat, with a depression at the center

—*lichen ruber planus*. Not rarely both varieties of papules occur together, and this is in favor of the view that both varieties are related and do not represent different diseases. The first papules are prone to appear upon the chest, the abdomen, the genitalia, and the flexor surfaces of the extremities. Gradually the papules become more numerous, and extend over large contiguous areas of skin. The skin is reddened and is covered with scales, which, however, never attain the thickness of those of **psoriasis**. The disorder differs also from **lichen of the scrofulous** in the freedom of the patient from scrofulosis, and from **eczema** by the fact that vesicles have not preceded the formation of scales. With the exception of the head, the mons veneris, the axilla, the **hair** of other parts of the body frequently falls out, but is generally replaced by thin lanugo. The **nails** become thin, brownish, fibrillated, and friable, while the palms of the hands and the soles of the feet become thicker and fissured. Often the remainder of the skin becomes thin and unyielding, so that the members assume an attitude of flexion, and can be but imperfectly flexed. Fissures also form readily. The patients often suffer from **nervous disturbances**, such as neuralgia, roaring in the ears, and sleeplessness. Occasionally papules develop also upon the **mucous membrane of the mouth**, appearing in the form of grayish spots, and sometimes undergoing superficial degeneration. Left to itself, lichen ruber induces progressive exhaustion and death, although arsenic has proved to be an efficacious remedy, so that the prognosis is not unfavorable.

Anatomic Alterations.—The papillæ of the cutis are infiltrated with round cells, and cellular proliferation can be demonstrated also in the Malpighian layer and in the epidermis. The blood-vessels of the cutis are at first dilated, but subsequently they are destroyed and their walls frequently undergo colloid degeneration. Lichen ruber planus is attended with flattening of the papillæ of the cutis in the situation of the depression. The external sheath of the root of the hair is often developed excessively and gives off lateral processes. Also, the erector muscles of the hair are greatly developed, while the lower terminations of the hairs are often fibrillated.

Etiology.—Nothing is known with regard to the etiology of lichen ruber. Experience has shown that the disease occurs most frequently in men between the sixteenth and the fortieth year of life.

Treatment.—**Arsenic** is properly considered a specific. Subcutaneous injections of solution of potassium arsenite (1 : 10) may be given advantageously. If itching be marked, the skin should be anointed twice daily with **carbolated ointment** (5.0 : 100).

II. SECRETORY DISORDERS OF THE SKIN.

SECRETORY DISORDERS OF THE SWEAT-GLANDS.

INCREASED SECRETION OF SWEAT (HYPERIDROSIS).

Increased secretion of sweat may occur either upon the whole of the body, or upon one side, or locally.

General increased secretion of sweat occurs especially in *obese persons*, but occasionally in cases of *epilepsy*, in advance of or following a convulsive seizure, or in place of such an attack; and also in the course of a number of *infectious diseases*: pulmonary tuberculosis, acute articular rheumatism, typhoid fever, during the *crisis* of febrile infectious diseases, and in cases of trichinosis. Profuse secretion of sweat often gives rise to *miliaria*. In obese persons engaged in active movement maceration of the skin and eczema intertrigo may readily occur upon opposed portions of the body. The most certain means of preventing general sweating consist in *cool ablutions with vinegar* [illegible] parts of vinegar to 1000 parts of water) and *atropin*.

℞ Atropin sulphate [illegible] gramme;
Powdered althea-root, sufficient to make [illegible]
Dose: 1 or 2 pills at night.

The skin should subsequently be dusted with a salicylated powder:

℞ Salicylic acid,
Zinc oxid, each, 10.0 (2½ drams);
Talcum, 20.0 (5 drams).—M.
Use as a dusting-powder.

Sweating or *hyperidrosis of the hands* is extremely annoying to the patient, because the moisture is deposited upon all objects with which the hands come in contact. Often the hands present a bluish discoloration and feel cool, so that in shaking hands with such a person one involuntarily draws back on contact with the amphibia-like wet and cold hand. Occasionally the epidermis is raised in places in blisters, or it becomes macerated and is exfoliated in shreds. Not rarely the condition is observed in pallid and nervous individuals. The *treatment* is the same as that for axillary sweating.

Sweating or *hyperidrosis of the feet* is a troublesome disorder that occurs with especial frequency in pallid and nervous persons. The sweat secreted readily undergoes decomposition, and gives rise to so disagreeable an odor (osmidrosis) that the presence of the disease can be readily detected at some distance by the sense of smell. The patients are often embarrassed in walking, as the skin becomes macerated, peels readily, and becomes fissured and sore. In mild cases of sweating of the feet daily *foot-baths* should be prescribed, *daily change of stockings*, and, after the bath, dusting the feet and the stockings with the *salicylated powder* previously mentioned. In marked cases, however, a layer of the *diachylon ointment* of Hebra should be applied upon strips of linen, with which the feet, and particularly the spaces between the toes, should be covered. The ointment should be renewed twice daily. In from six to twelve days the horny epidermis is exfoliated, and a healthy epidermis comes into view. As a precautionary measure daily foot-baths should be continued, and the feet should be dusted with salicylated powder.

DIMINISHED SECRETION OF SWEAT (ANIDROSIS).

Diminished secretion of sweat occurs in cases of *diabetes, polyuria*, and *contracted kidney*, because the body loses a large part of its water through the urine. The condition is often observed after *long-continued debilitating diseases*, as, for instance, *carcinoma* and *chronic diseases of the skin* (eczema, psoriasis, pruritus, lichen, ichthyosis, and leprosy). Occasionally *nervous influences* are operative; thus, sweating is at times absent from paralyzed members. The condition is without especial significance.

ALTERATIONS IN THE CHARACTER OF THE SWEAT (PARIDROSIS).

Osmidrosis or **bromidrosis** signifies an alteration in the odor of the sweat. Often an unpleasant odor is present, and this may be developed after the sweat has been secreted, as has been mentioned in the consideration of sweating of the feet. Occasionally in cases of hysteria and in nervous persons the sweat has an odor of musk or of violets.

Chromidrosis signifies an alteration in the color of the sweat. In the presence of *jaundice* the sweat contains biliary coloring-matter, and imparts a yellow color to the linen. Occasionally blue sweat (cyanidrosis) is observed, usually appearing upon circumscribed portions of the body, as, for instance, the eyelids. Little of a definite nature is known with regard to the blue pigment. Recently there has been a tendency to consider the pigment as an indigo-body. Further, chromogenic bacteria also may cause blue sweating.

The sweating of blood—hemidrosis—is scarcely discussed appropriately in this place, for the condition is one of undue friability of the blood-vessels, in consequence of which the extravasated blood finds its way into the sudoriferous glands or into their immediate vicinity upon the surface of the skin.

Uridrosis is the best-known example of abnormal composition of the sweat. It is encountered in cases of scarlet fever and of cholera-nephritis if the secretion is suppressed and urea is excreted from the blood through the sweat. Should sweat rich in urea evaporate upon the skin, a whitish deposit of crystalline urea remains behind.

SECRETORY DISORDERS OF THE SEBACEOUS GLANDS.

INCREASED SEBACEOUS SECRETION (SEBORRHEA).

Increased sebaceous secretion is known also as *seborrhea,* of which two varieties, the *oily* and the *dry,* have been distinguished, accordingly as the skin presents a glistening, greasy appearance or is covered with crusts of fat (oily seborrhea) or with desquamated epithelial cells admixed with fat-drops (*dry seborrhea*). In accordance with the distribution of the disease a distinction is made between universal and local seborrhea, and of the two the latter especially is of practical significance.

Seborrhea of the scalp occurs principally in infants, and is frequently attended with an accumulation of grayish-yellow or grayish-green crusts upon the scalp, which have a disagreeably rancid odor, feel greasy, and consist of masses of fat and epidermic cells. If the crusts are detached, unaltered skin comes into view. Less commonly the masses of fat have undergone decomposition and have excited irritation of the skin, as indicated by redness. Left to itself, spontaneous recovery takes place toward the close of the second or during the third year of life, the crusts becoming brittle, breaking up and being cast off by the growing hair. The condition can be recognized, and is distinguished from *eczema* by the fact that the latter condition is not confined to the boundary of the hair, and gives rise to redness and weeping of the skin. Confusion with *psoriasis* is scarcely possible, as this disorder does not occur in infants, and, besides, it gives rise to plaques of psoriasis upon the skin. *Treatment* is often permitted by the parents only with opposition, because they consider seborrhea of the scalp as a curative condition whose removal might be attended with danger. The head should be generously anointed morning and evening with *olive-oil*, and, besides, a strip of flannel that has been dipped in the oil should be applied to the crusts day and night. When the crusts have become detached the head should be washed with *green soap*. Then the inunction and the washing should be persisted in for several days. In *adults* seborrhea of the scalp occurs most commonly as a *dry seborrhea*, and is known also as *pityriasis of the scalp*. It consists in the formation of an abundance of scales of skin containing fat, and which in part accumulate between the hairs, but in part drop upon the clothing. The hairs generally fall out, and baldness thus results—*pityroid* and *furfuraceous alopecia*—which, however, disappears with the relief of the primary disorder. Among the *causative factors*, anemia, antecedent disease of the skin (eczema, erysipelas, small-pox), syphilis, menstrual disorders, pregnancy, and parturition have been mentioned. The *treatment* consists in washing the scalp in the evening with *alcohol* and inunctions of *olive-oil* in the morning. In addition the fundamental disorder (anemia, syphilis) should be relieved.

Seborrhea of the face is generally of the oily variety, and is attended with a fatty, glistening appearance, especially of the forehead, the nose, and the chin, and giving rise to spots of fat when touched with blotting-paper. Upon the nose in particular the orifices of the sebaceous glands appear dilated and discolored gray or black by dust. The disorder occurs especially in anemic persons at the period of puberty. On the eyebrows pityriasis of the hair may develop. The *treatment* is the same as that for seborrhea of the scalp.

Seborrhea of the Genitalia.—In males seborrhea occurs especially in the presence of a contracted prepuce—*phimosis*. Under such circumstances the preputial sebaceous matter is converted into a crust, which finally encloses the entire gland, occasionally undergoes calcification, and gives rise to the formation of *preputial calculi*.

In walking and during the summer decomposition of the smegma readily takes place, and the decomposed secretion causes irritation of the prepuce and of the glans and excites a balanitis or a balanoposthitis. Under such conditions excoriations, adhesions between the prepuce and the glans, and acuminated condylomata may occur. The marked irritation and itching readily induce onanism. Slight degrees of phimosis should be corrected by retraction of the prepuce upon the glans several times daily, preferably after the evacuation of urine, and maintaining the retraction for some time. More marked degrees of phimosis must be relieved by operative means. The accumulated crusts of fat should then be anointed every two hours with *olive-oil* until they are exfoliated, the glans and the prepuce be washed with *green soap*, and then *lead-water* be applied. In *girls* and *women* accumulations of smegma take place upon the clitoris and between the labia, and will require the same treatment as the analogous condition in males.

Universal seborrhea occurs occasionally in the newborn, the active secretion of sebaceous matter that during fetal life gives rise to the formation of the caseous vernix persisting during the first days of life. The skin is covered with large fatty scales, so that it appears as if plowed. The condition is therefore designated also *sebaceous ichthyosis*, or *cutis testea*. Often the temperature of the body is abnormally low, food is rejected, and collapse occurs, and may lead to death. The children should be given *milk* at regular intervals, should be placed twice daily in a *warm bath* at 42.5° C. (34° R.—108.5° F.), and the skin after the bath should be anointed with *olive-oil* in order to remove the scales. In bed *hot bottles* should be placed at the side of the body of the child. In *adults* universal seborrhea occurs especially in the sequence of debilitating diseases. The skin becomes rough and covered with grayish scales, and the condition is designated *pityriasis tabescentium*, *scrofulosorum*, *carcinomatosorum*, etc., in accordance with the nature of the primary disease. Inunctions of the skin with oil will then render it again pliable and smooth.

DIMINUTION IN THE SEBACEOUS SECRETION (OLIGOSTEATOSIS).

Diminution in the sebaceous secretion is attended with dryness of the skin and a tendency to fissures and the formation of scales. When the skin is covered with epidermic scales the condition is designated also *simple pityriasis*. The disorder may be *congenital* or *acquired*, and develops particularly in the course of a number of chronic diseases of the skin, as, for instance, ichthyosis, psoriasis, and prurigo. Locally it occurs often upon the dorsum of the hands in persons who frequently wash them, particularly during the winter; under such circumstances "chapped hands" result.

The disorder can be relieved by inunctions of the skin with *oil*, *wool-fat*, or *glycerin*. In winter *gloves* should be worn.

DISORDERS OF SEBACEOUS SECRETION (PARASTEATOSIS).

COMEDO.

Symptoms and Diagnosis.—Comedones consist in **black dots** upon the skin, situated in the orifices of the sebaceous follicles, and often projecting somewhat above the level of the skin. If lateral pressure be exerted, the black dot escapes externally, and is followed by a spiral yellow thread, resembling a tiny worm, which consists of inspissated sebaceous matter, and on microscopic examination will be found to contain fat-drops, glandular cells involved in fatty degeneration, cholesterin, leucin, crystals of tyrosin, and, not rarely, acarus of the follicles and bacteria. Comedones are often present in such large number and so closely together that the skin presents a densely mottled appearance. The skin is sometimes elevated like a disc, or a wart, or a nipple. Comedones are especially numerous upon the forehead, the nose, the lips, the chest, and the back, because sebaceous follicles are present in large number in these situations. Comedones are often associated with acne.

Etiology.—Comedones develop with especial frequency at the *period of puberty* in **anemic individuals**, perhaps in consequence of deficient activity in the forces that bring about excretion of the sebaceous matter. Occasionally the condition is the result of **mechanical obstruction** of the orifices of the sebaceous follicles, such as occurs in laborers in tar-works and in oil-works. Sebaceous matter of excessive density also appears to be responsible for the development of comedones.

Prognosis and Treatment.—Comedones are troublesome on account of the disfigurement to which they give rise, but otherwise they represent a wholly innocuous condition, which can be removed by **expression between the finger-nails** or with the aid of a **watch-key**. **Green soap** may be employed to wash the skin, in order to bring about increased exfoliation of the epidermis.

CUTANEOUS SAND (MILIUM).

Milia appear in the form of bright white nodules rather larger than a poppy-seed, over which the epidermis is stretched. If they be incised with a knife, hard, crumbling matter can be expressed containing epidermis-cells arranged like the layers of an onion, but generally without fatty degeneration. These masses have collected in a lobule of a sebaceous follicle, or within the entire glandular space. Milia are especially common upon the eyelids, the cheeks, and the genitalia. In the coronary sulcus of the glans they are occasionally present in large number, lying close together. They develop with especial readiness at the periphery of *cutaneous cicatrices;* further, in conjunction with *other diseases of the skin,* as, for instance, after pemphigus. Occasionally the condition appears to depend upon an abnormal composition of the sebaceous matter. The disorder can be readily relieved by *incision with the knife* and *removal of the contents by pressure.*

III. HYPERTROPHIES OF THE SKIN.

HYPERTROPHIES OF THE PIGMENT OF THE SKIN.

NEVUS (MOTHER-MARK).

The designation mother-mark is applied to yellowish, brownish, or even blackish discolorations of the skin that are congenital, but frequently increase progressively in size after birth. On microscopic examination brownish and blackish pigment-granules are found in the lowermost cells of the Malpighian layer, in the cells of the cutis, and often also in obliterated vessels of the cutis. The pigment gains entrance into the cells of the Malpighian layer from the cutis. Mother-marks with a smooth covering of epidermis are designated *nevi spili;* those with a nodular surface, as *verrucose nevi;* and those covered with hair, as *hairy nevi.* Occasionally they are attached to the skin by a pedicle like a tumor, and are then designated *molluscitorm nevi.* They vary greatly in number and size. Occasionally they are so densely packed together that the skin presents a spotted appearance. At times they follow the course of the nerves upon one or both sides —*nerve-nevi*—and among other structures surround the gluteal and the pubic region like bathing-trunks. Occasionally nevi become carcinomatous at a later period. Their removal can be effected only by *operative means.*

CHLOASMA.

The designation chloasma is applied to *acquired discolorations of the skin* which give rise to a brownish tint. In accordance with the causative factors, several varieties of chloasma are distinguished.

Freckles, ephelides, or **lentigenes** are roundish, brownish spots which appear especially upon the nose, the adjacent cheek, and forehead, but also upon covered portions of the body (flexor surfaces of the extremities, the genitalia), and therefore are due not alone to the action of the sun. Freckles usually develop in especially large number in blond and red-haired individuals with a delicate skin. Generally they do not appear before the sixth or after the fortieth year of life. Most frequently they develop at the period of puberty, disappearing spontaneously subsequently. When present in large number they give rise to disfigurement. They can be removed by means of agents that induce active exfoliation of the skin, as, for instance, frictions with *green soap*, applications of *tincture of iodin* or of a solution of *mercuric chlorid* (0.5 : 50). When present in large number they may be concealed by means of cosmetics.

Traumatic chloasma is attended with dark discoloration of the skin at places where an injury has been inflicted, as, for instance, by the pressure of belts or bands, as a result of scratching for relief from itching cutaneous eruptions, irritating poultices and plasters (mustard-plaster, cantharidal plaster). When pediculi have been present in the clothing for a long time the skin of the trunk especially acquires an almost black color, and this is known as melasma (melanoderma, nigrities), or, if desquamation takes place, as pityriasis nigra.

Caloric chloasma is the designation applied to those dark discolorations of the skin that result from the action of the rays of the sun.

Uterine chloasma occurs especially upon the forehead and the cheeks in women suffering from menstrual disorders or uterine or ovarian disease. The chloasma of pregnancy, which develops in gravid women, also belongs in this category.

Cachectic chloasma occurs in cases of pulmonary tuberculosis, carcinoma, malaria, and syphilitic cachexia.

Dark discoloration of the skin is the principal symptom of **Addison's disease.**

From the presence of *abnormal pigments* discoloration of the skin occurs in cases of jaundice; after the use of silver nitrate in the form of *argyria;* and after the administration of arsenic in the form of *arsenical melanosis.* Blackish discoloration, particularly of the hands and the forearms has been observed in workers in silver, from the penetration of *silver-dust*, in stonecutters and millers from iron-dust—*siderosis of the skin*, and in chimney-sweeps and firemen from coal-dust—*anthracosis of the skin.*

HYPERTROPHY OF THE EPIDERMIS (KERATOSIS).

ICHTHYOSIS.

Etiology, Symptoms, Diagnosis, and Prognosis.—Ichthyosis is generally an **hereditary disease.** Cases that are not hereditary are extremely rare, and then occur usually in connection with other antecedent disease of the skin. In the last instance the keratosis does not involve the entire skin (total ichthyosis), but only circumscribed portions (partial ichthyosis). Also in the hereditary cases the earliest alterations in the skin do not appear until *after the first year of life.* In the mildest form of the disease the skin exhibits a tendency to desquamation in large quadrangular areas. At the same time the skin is dry and exceedingly rough—*simple ichthyosis.* Not rarely the borders of the individual cutaneous scales appear slightly raised, while the center is depressed—*scutellate ichthyosis.* If the individual areas present a mother-of-pearl appearance, especially toward their free borders, the condition is designated *nitid ichthyosis.* At times the epidermic scales present a grayish discoloration, so that the appearance of the skin is suggestive of that of the skin of a snake—*serpentine* or *cyprine ichthyosis.* In the most marked cases the epidermic scales appear horny, thickened, and curved—*corneal ichthyosis;* or the epidermis is the seat of thorny, grayish-black projections—*histricine ichthyosis.* Patients of the latter kind, who are occasionally exhibited in museums as curiosities, have also been designated *porcupine-people.* Often the different portions of the skin are affected in varying degrees.

The earliest changes in the skin are observed upon the **extensor surfaces of the elbows and the knees.** The flexures of the joints, the axillary cavities, the palms of the hands, the soles of the feet, and the genitalia remain uninvolved. The face and the scalp also are generally but little affected, although they may desquamate freely. Frequently there is marked loss of hair. Patients with ichthyosis perspire little, complain frequently of distressing itching of the skin, and occasionally suffer from painful fissures. At times hypertrophy of the heart, albuminuria, or polyuria has been observed. The disease generally persists throughout the whole of life, although remissions and exacerbations frequently occur. Life is not endangered. Improvement may be effected temporarily. In isolated instances recovery has taken place spontaneously, as, for instance, after recovery from small-pox.

Anatomic Alterations.—Ichthyosis is attended with excessive proliferation of the cells of the **epidermis.** In the prickle-layer of the epidermis the marked development of the prickle-processes and the thickness of the intercellular tissues are conspicuous. Processes of division and multiplication also are present in some of the cells. In addition, black pigment occurs, partly within and partly outside of the cells. Proliferation and prolongation of the papillary bodies are encountered in the **cutis.** The blood-vessels are dilated and the entire cutis is unusually dense. The sudoriferous and the sebaceous glands often are atrophied. The **subcutaneous connective tissue** is distinctly atrophied.

Treatment.—Temporary improvement and even cure can be effected by bringing about active desquamation of the epidermis. To this end the skin should be rubbed at night with **green soap**, and a tepid bath (35° C.—28° R.—95° F.), to which 150 grams of soda are added, should be taken in the morning for an hour, and the body should be anointed with **carbolated ointment** (carbolic acid, 5.0—75 grains; wool-fat, lard, each, 25.0—1¼ ounces). The procedure must be repeated daily for weeks.

ACANTHOSIS NIGRICANS.

Acanthosis nigricans is a *rare disorder of the skin,* which occurs with relative frequency in patients suffering from *carcinoma.* The diseased areas of skin present a brownish, grayish, or blackish *discoloration,* and besides are *verrucose* and *nodular,* as if covered with numerous small warts or broad condylomata. The nape of the neck, the axillary region, the fold of the breast, the umbilicus, the genitalia and the adjacent thigh, the rectum and the perineum are affected with especial frequency. Also the *mucous membranes* of the mouth and the pharynx, the lips, and the nose, share in the alteration and exhibit numerous grayish, pigmented warts. *Anatomically,* proliferation of the papillæ of the cutis, dilatation of the blood-vessels and the lymphatics, round-cell accumulation in the vicinity of the vessels, pigment in some of the cells of the cutis, proliferation of the prickle-layer of the epidermis, and pigment-granules in the cells of the epidermis are present. *Treatment* is powerless, for removal of the diseased skin with the knife is contraindicated on account of the generally extensive distribution of the disease.

HYPERTROPHY OF THE HAIR (HIRSUTIES).

Excessive growth of hair may be congential or acquired, and accordingly a distinction is made between *congenital* and *acquired hirsuties.*

Congenital hirsuties is manifested in some persons by the presence at birth of unusually abundant and long hair upon the scalp and lanugo, but which soon fall out. At times the entire body is densely covered with hair, so that such individuals have been designated hairy persons. The disorder is *hereditary* in some families and is often associated with *deficient development of the teeth.* Further, the condition is not actually one of hypertrophy of the hair, but it is a developmental defect. Not rarely *nevi* are covered with an abundance of often thick hairs, which at times do not appear until after birth. Thick and not rarely long masses of hair occur in cases of *occult bifid spine,* generally in the lower portions of the spinal column, and should suggest to the clinician

Acquired hirsuties is attended, among other conditions, with vigorous development and long growth of the hair of the head, the beard, the axilla, and the mons veneris, and in the aged it involves also the hair of the nose and the ears. In the same category belong the development of a beard in women and abnormal growth of hair in paralyzed members and on cicatrices. The disorder is often a most disfiguring one, from which temporary relief can be afforded by removal of the hairs or *depilating remedies;* but permanent relief can be secured only by *destruction of the hair-follicles* by means of caustic pastes or the galvanocautery. The *Roentgen rays* also have recently been employed as a depilatory.

HYPERTROPHY OF THE NAILS (ONYCHAUXIS).

Hypertrophy of the nails is characterized by thickening of the nails, and frequently also by excessive growth in width and length, so that the cracked and brittle nail becomes curved like a claw—onychrogryphosis. Often the matrix of the nail becomes inflamed—paronychia. The condition frequently results from *pressure,* but it occurs also in association with *chlorosis* and *pulmonary tuberculosis,* and can be corrected only by *surgical means.*

Induration of the connective tissue in the newborn—sclerema of the newborn.—Induration of the connective tissue in the newborn occurs, as the name of the disease indicates, generally in infants within the first few days or weeks of life, and but rarely at a later period. Often the children have been asphyxiated at birth or were attacked by pneumonia or gastro-intestinal catarrh. The first alterations appear in the calves, whence they extend to the thighs, the trunk, the arms, and the face. The *skin* becomes reddened and is slightly edematous, but subsequently it becomes peculiarly firm and rigid. At the same time the *bodily temperature* declines (occasionally to 22° C.—71.6° F.). The child becomes apathetic and death frequently results amid symptoms of collapse. Occasionally improvement temporarily occurs, recovery but rarely. Possibly the deficient circulation of blood in the skin is the cause of the disorder, so that the subcutaneous connective tissue, which in the newborn contains an abundance of solid fatty acids (palmitic, stearic), becomes rigid. The assumption of a bacterial origin for the disease is as yet only hypothetic. In treatment, *baths* at a temperature of 37° C. (98.6° F.) should be prescribed thrice daily for half an hour, the body should be surrounded by *hot-water bottles,* milk, meat-broth, or dilute wine should be administered with a teaspoon, or the *skin* should be *massaged* carefully morning and evening. In the presence of threatening collapse subcutaneous *injections of camphorated oil* should be resorted to.

IV. ATROPHY OF THE SKIN.

ATROPHY OF THE PIGMENT OF THE SKIN (LEUKODERMA).

Atrophy of the pigment of the skin is indicated by the formation of *whitish spots* upon the skin. If the condition is congenital it is designated albinism, while if it is acquired it is designated vitiligo.

Albinism either involves the entire skin or appears in circumscribed areas. In cases of **universal albinism** the skin not only presents a bright white appearance, but the hairs are flaxen and the reflex from the fundus of the eye is red in consequence of a deficiency of pigment in the iris and the choroid. Such persons are designated albinos. The condition occurs with especial frequency in some countries, such as Sicily, and is hereditary in some

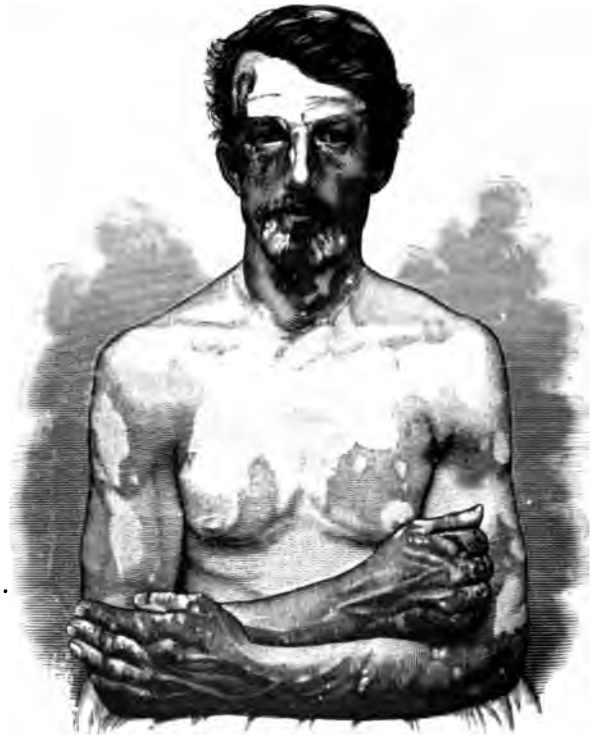


FIG. 33.—Vitiligo with involvement of the hair of the head; from a photograph (personal observation, Zurich clinic).

families. **Partial albinism** occurs especially upon the genitalia, the dorsum of the hands and the feet, the face, and the scalp. Generally the hairs in the area of the white spots also present a bright white color. The disorder is especially disfiguring in negroes, who present a spotted appearance and are known also as magpie-negroes.

Vitiligo develops after *emotional disturbances*, in *the course of nervous diseases* (*exophthalmic goiter*), *debilitating diseases* (*typhoid fever, syphilis*), and *traumatism*, and occurs almost always in *adults*. Occasionally the margins of the white spots present a brownish tint, or a brownish area is present at the center of the spots, so that an impression is made as if the pigment of the skin had withdrawn to the periphery or the center of the spot. Such hairs as may be present take part in the light discoloration (p. 204, Fig. 33). At times the spots are distributed symmetrically upon both sides of the body, and they may follow the course of the cutaneous nerves. All of the pigment-atrophies of the skin are only disfiguring affections, for the relief of which nothing can be done beyond concealing them by means of *cosmetics*.

ATROPHIC CHANGES IN THE HAIRS.

GRAYING OF THE HAIR (CANITIES).

Graying of the hair occurs in advanced life—*senile canities*—because the hair-bulb produces but little pigment and the cortex of the hair becomes deficient therein. Accordingly, the graying of the hair takes place from below upward. The hair in the temporal region generally becomes gray earliest, and subsequently that of the remainder of the head and the beard. The designation *premature canities* is applied to graying of the hair before the thirtieth year of life. This is hereditary in some families, but occurs also after alcoholic and venereal excesses, and develops occasionally after severe infectious disease (typhoid fever), after hemicrania and neuralgia of the nerves of the head, and also as a result of anxiety, care, and excessive mental activity. Profound emotional disturbances are believed to cause graying of the hair within a few hours. The deficiency of pigment in the hair in cases of *albinism* and *vitiligo* has already been mentioned in the preceding section. To conceal gray hair various *dyes* have been recommended, including silver nitrate, pyrogallic acid, and powdered henna.

DEFICIENCY OF HAIR (ATRICHIA; LOSS OF HAIR; ALOPECIA).

Congenital atrichia is rare, and is generally associated with deficiency of the teeth and the nails. Occasionally, however, the hair may appear in the second or the third year of life. *Acquired* *atrichia* occurs at times as the result of nervous disturbances, as, for instance, after cerebral concussion. Complete loss of hair takes place occasionally also in persons who have been struck by lightning. At times, however, the disorder has appeared without demonstrable cause. The loss of hair generally begins upon the scalp, then extends to the beard, the eyebrows, and the eyelashes, also to the axilla and the mons veneris, until finally the patient is wholly deprived of hair.

Loss of hair—defluvium capillorum—is a natural occurrence in infants and the aged, although immediate restitution takes place in the former, while in the latter the hair is not regenerated, because the process is dependent upon senile involutional processes in the papillæ of the hair, including, among other conditions, obliteration of the blood-vessels. The loss of hair occurs earliest at the vertex, where it gives rise to the well-known bald pate, or at the junction of the forehead with the scalp, whence it extends to the temporal region. The beard generally is not affected. *Premature loss of hair* is hereditary in some families. Occasionally it appears to be a result of excessive mental activity. A dissipated mode of life also is properly considered an etiologic factor. At times it occurs in the sequence of severe disease, as, for instance, typhoid fever, carcinoma, pulmonary tuberculosis, or syphilis, although the hair generally grows again. It often occurs after parturition. Occasionally loss of hair is a result of disease of the scalp, and should the papillæ of the hair under such circumstances be themselves diseased, regeneration of the hair cannot be expected. The hair often falls out in the presence of seborrhea of the scalp—so-called furfuraceous or pityroid alopecia; but the hair not rarely falls out also in connection with eczema, psoriasis, favus, herpes tonsurans, lichen ruber, lupus, and after erysipelas of the scalp.

In the **treatment** of baldness, **spirituous applications** should be made to the scalp, with subsequent **inunction, as, for instance:**

℞ Dilute alcohol,	100.0 (3¼ fluidounces);
Carbolic acid,	2.0 (30 grains).—M.

To be rubbed in morning and evening.

℞ Oil of mace,	10.0 (2½ fluidrams);
Olive-oil,	40.0 (10 ").—M.

To be rubbed into the hair.

CIRCUMSCRIBED ALOPECIA (ALOPECIA CELSI).

Circumscribed alopecia is attended with loss of hair in circular, but otherwise unaltered areas of the scalp (p. 207, Fig. 34), less commonly also of the beard. The areas gradually increase in size, become confluent, and eventually almost all of the hair of the head may be lost. Often the areas exhibit a symmetric distribution upon the two sides of the head. Occasionally *nervous symp-*

toms are present, especially mental depression and sleeplessness. Anesthesia also has been said to be present in the bald areas. At times there has been inequality of the pupils. The disorder generally pursues a *chronic course,* persisting at times for several years, and then not rarely exhibiting remissions and exacerbations. Generally the hair grows again, but often it is at first lighter and rather resembles lanugo. Nothing of a definite nature is known with regard to the *etiology* and the *nature of circumscribed alopecia.* By some observers the disorder is considered a *trophoneurosis,* and emphasis is placed upon the fact that it occurs in anemic and nervous persons and develops after emotional disturbances. Others attribute it to *parasitic influences,* laying emphasis especially upon its epidemic occurrence and infection in schools and barber-shops.

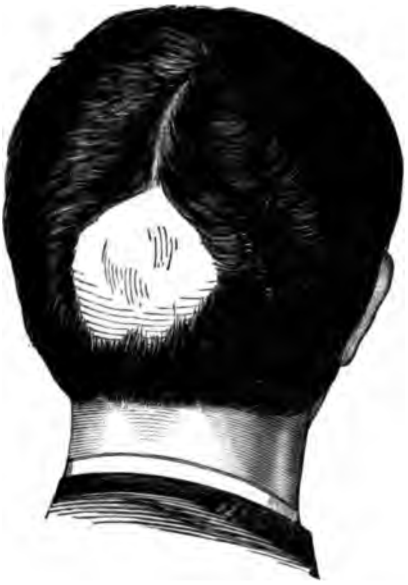


FIG. 34.—Circumscribed alopecia in the occipital region in a man 27 years old (personal observation).

The **treatment** consists in applications of *mercuric chlorid* (1 : 1000) to the head, followed by *inunctions of oil* (oil of mace 10.0—2½ fluidrams, olive-oil 40.0—1¼ fluidounces).

BRITTLENESS OF THE HAIR (TRICHORRHEXIS).

Long hairs upon the head and in the beard occasionally undergo fibrillation at their extremity—so-called trichoptilosis—for the relief of which the only remedy consists in cutting the hair with scissors. Occasionally, small nodules form upon the hair of the beard at short intervals, like the ova of pediculi, and at times also upon the eyebrows and the pubic hair—so-called *nodose trichorrhexis.* On traction the hairs readily break at these points. On microscopic examination it will be found that the cortical cells of the hair are fibrillated in the situation of the nodes and project into one another like hair-brushes (Fig. 35). Often such alterations probably result from too vigorous rubbing in the process of drying; whether parasitic influences are also operative has not been demonstrated.

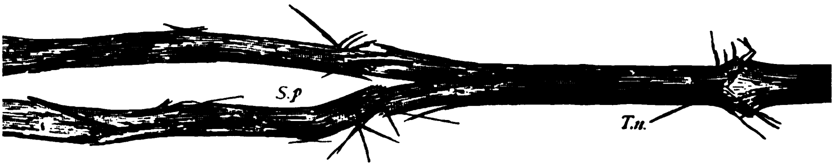


FIG. 35.—Splitting of the end of a hair: *T.n.*, trichorrhexis nodosa; *S.p.*, scissura pilorum (Michelson).

In South America nodular formations of brownish color upon the hair have been described and designated *piedra*, and the cause for which has been demonstrated to be a *filamentous fungus. Collections of micrococci* occasionally give rise to the formation of nodules upon the hairs. At times atrophy of the medulla and the cortex of the hairs takes place in limited areas, so that the intervening healthy portions of hair present a spindle-shaped enlargement, and the condition has been designated *intermittent* or *moniliform aplasia of the hair.*

ATROPHIC DISORDERS OF THE NAILS (ONYCHOATROPHY).

Congenital atrophy of the nails occurs occasionally in conjunction with deficient development of the fingers, the toes, and the teeth. The nails may be totally wanting or be but imperfectly developed. *Acquired atrophy of the nails* develops in the sequence of *severe diseases*, such as pulmonary tuberculosis, typhoid fever, carcinoma, and diabetes. Under such conditions the nails become thin and dark-colored and they peel. *Chronic diseases of the skin* also (eczema, psoriasis, ichthyosis, lichen ruber) are occasionally followed by atrophy of the nails. At times white spots appear in the nails in consequence of the presence of air. Such a condition has been observed in the sequence of typhoid fever and of polyneuritis, but sometimes no cause could be demonstrated.

ATROPHY OF THE CUTIS.

Among the circumscribed atrophies of the skin are the so-called *striæ*, which develop after marked stretching of the skin. The cicatrices of pregnancy are the best known, but striæ appear upon the abdominal walls also after peritonitis, ascites, ovarian and other abdominal tumors. They appear upon the thighs and the buttocks in obese and edematous individuals and as a result of nervous influences. *Senile atrophy of the cutis* occurs at advanced age, and gives rise to thinning and wrinkling of the skin. Occasionally *diffuse atrophy of the skin* develops in early life. This generally begins in the lower extremities, and extends thence to the trunk and the arms. The skin becomes thin and dry and appears wrinkled like cigaret-paper. It can be readily raised in folds, and is covered with brownish and reddish spots, so that it presents a mottled appearance, as from contusions. Under such conditions all of the layers of the skin have been found atrophied. The disorder should not be confounded with *relaxed skin*, in the presence of which the patient is capable of lifting the skin in such enormous folds as will wholly enclose an extremity. Under these conditions mucoid transformation has been found in the cutis. The designation *simple xeroderma* has been applied to a form of congenital atrophy of the skin that begins in earliest childhood, and generally involves the lower extremities, much less frequently also the arms. The skin of the affected parts is thin and shiny, like gold-beaters' skin, often wrinkled and desquamating readily. It can be raised in folds only with difficulty, is exceedingly sensitive, and interferes with walking and prehension. The greatest relief is afforded by *oily inunctions*, in order to maintain the skin in a pliable condition. *Pigmented xeroderma* also generally begins in the first years of life, and occurs frequently in Jews. Occasionally it has been observed in several members of the same family. Uncovered portions of the body, especially the face, the ears, the nucha, the back, the forearms, the legs, and the feet, are first affected. Brownish and blackish spots appear upon the skin, which is thin and can be raised in folds only with difficulty. The intervening skin generally is remarkably white, but contains dilated vessels. In the course of from one to four years papillary elevations appear upon the skin, representing carcinomata, sarcomata, or angiomata. Neoplasms occasionally develop also in internal viscera. Death results in consequence of progressive exhaustion, as treatment (arsenic, removal of the cutaneous growths with the knife) has hitherto been unsuccessful.

V. CUTANEOUS NEUROSES.

ITCHING OF THE SKIN (CUTANEOUS PRURITUS).

Symptoms, Diagnosis, and Prognosis.—The designation **cutaneous pruritus** is applied only to such cases of itching of **the skin as are unattended** with alterations in the skin and are independent of other diseases of the skin. The itching is generally aggravated by heat, and is therefore especially marked at night, preventing sleep, and consequently inducing serious debility. Generally the skin is covered with scratch-marks and cicatrices, and if the disease has existed for a considerable length of time the skin acquires a brownish color and infiltrated character, in consequence of transformation of the hemoglobin of extravasated red blood-corpuscles and inflammatory irritation of the skin. The *duration of pruritus* depends upon the removability of the causative factors. The *prognosis*, likewise, is governed accordingly, for although itching of the skin is of itself an annoying rather than a dangerous disorder, it is, however, not rarely dependent upon the presence of incurable diseases. Exasperating pruritus has occasionally led to suicide.

Etiology.—Cutaneous pruritus occurs not rarely in advanced life—*senile pruritus*—and is then probably dependent upon involutional processes in the cutaneous nerves. Frequently it is the result of **auto-intoxication.** It occurs, therefore, in association with jaundice, diabetes, nephritis, carcinoma, gastric, hepatic, and uterine disease, and during pregnancy. Some patients are attacked by pruritus on the approach of winter—*hiemal pruritus*—with regard to whose mode of origin nothing is known. Occasionally pruritus occurs as an **hallucination,** as, for instance, in individuals that believe themselves the victims of cutaneous parasites.

Treatment.—For the relief of cutaneous pruritus **inunctions of carbolated ointment** particularly may be recommended:

℞ Carbolic acid,	5.0 (75 grains);
Wool-fat,	
Lard,	each, 25 (6 drams).—M.

For inunction twice daily.

Friction with lemon-juice or **ablutions with dilute vinegar** occasionally afford great relief. Internally potassium bromid may be administered:

℞ Potassium bromid,	10.0 (2½ drams);
Extract of belladonna,	0.3 (4½ grains);
Carbolic acid,	1.0 (15 ");

Extract of licorice, sufficient to make fifty pills.—M.
Dose: One pill four times daily.

Sodium salicylate and **salophen** (each 1.0—15 grains—every two hours) also have been recommended. In addition, the primary disorder should be treated.

VI. PARASITES OF THE SKIN (PARASITIC DERMATOSES).

ANIMAL PARASITES OF THE SKIN (DERMATOZOÖNOSES).

ITCH (SCABIES).

Etiology and Anatomic Alterations.—The symptoms of scabies are due to the **itch-mite,** *Acarus scabiei,* or *Sarcoptes hominis.* Healthy individuals acquire the parasite either through intimate association with a patient suffering from scabies, or through the use of articles of linen or clothing that had been previously worn by such a patient and had become thereby infected with the itch-mite, or, finally, through infection from animals, for

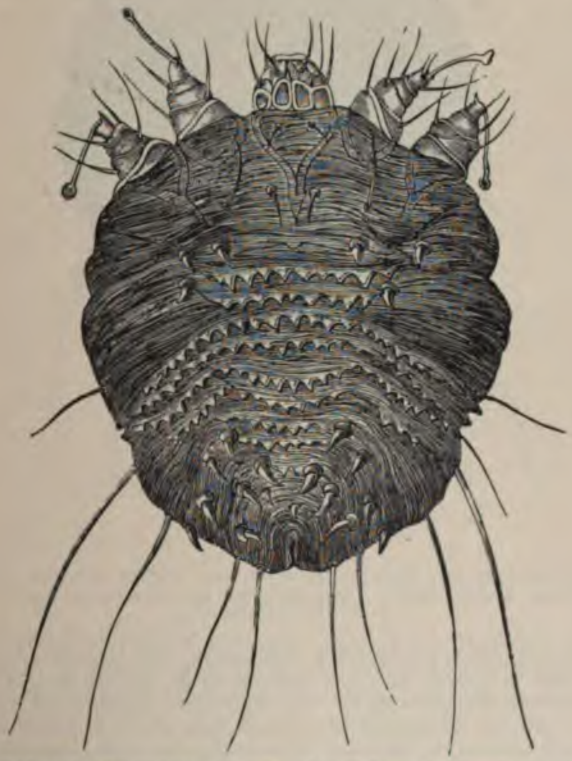


FIG. 36.—Female, sexually mature itch-mite, viewed from the dorsal aspect; magnified 300 times (after Kaposi).

so-called mange in cats, dogs, horses, and cows is often only scabies. Transient association with a patient suffering from scabies, as, for instance, in medical examination, will not necessarily give rise to infection; but this frequently occurs among those who *sleep together*. Also cheap lodging-houses, in which the bed-linen is changed but seldom, constitute a frequent source for infection.

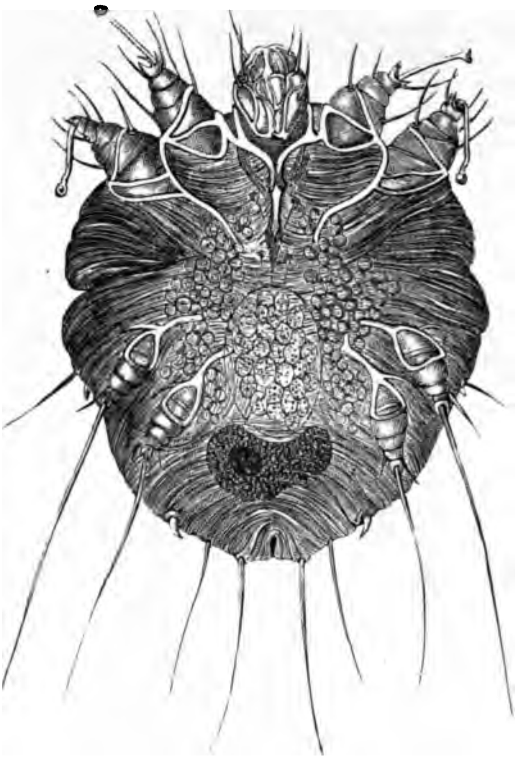


FIG. 37.—Female, sexually mature itch-mite, viewed from the ventral aspect; magnified 300 times (after Kaposi).

The *itch-parasite* is just visible to the naked eye as a small, grayish-white dot. Its form suggests that of a tortoise (Figs. 36, 37, 38). The males, whose number is considerably less than that of the females, are smaller than the latter, and can be readily recognized from the bifurcated penis upon their ventral aspect. In addition, they possess not only upon the two fore-feet, but also upon the hind feet, a sucker (ambulacrum), while the two pair of hind feet of the female possess bristles. After fertilization the female bores its way into the epidermis, and forms the *itch-groove*, where it deposits its ova, and from these the larvæ develop in from six to twelve days. The larva at first possesses but three pair of feet. It exfoliates three times, and emerges from the first exfoliation with four pair of feet. The female parent deposits on an average two eggs daily, and after a single fertilization deposits as many as fifty eggs altogether.

Symptoms and Diagnosis.—In the clinical picture of scabies two sets of symptoms must be differentiated, namely, the alterations to which the itch-mite itself gives rise, and, besides, the changes in the skin resulting from the itching and the scratching. The latter are, without doubt, the more extensive. The changes of the first variety are confined essentially to the boring of an itch-groove in the epidermis on the part of the fertilized and pregnant female acarus. The males, apparently, remain in depressions in the skin close to the orifices of the itch-grooves. The length of such a groove occasionally reaches 5 cm., although generally it is from 0.5 to 1 cm. Each female strives to bore more and more deeply, in order to obtain new, juicy, nutritive material. After the act of parturition has been completed the female parent dies at the deepest portion of the groove. An itch-groove can be readily recognized from its sharp limitation, inasmuch as it acquires a black appearance from the presence of dirt. Its course is generally tortuous. If it be punctured with a needle, the parasite can be readily extracted. Frequently, the entire itch-groove, together with its contents, can readily be removed from the epidermis by means of scissors, and be examined with the microscope, particularly if it be cleared by the addition of potassium hydroxid (p. 214, Fig. 39). Often the patient has scratched open

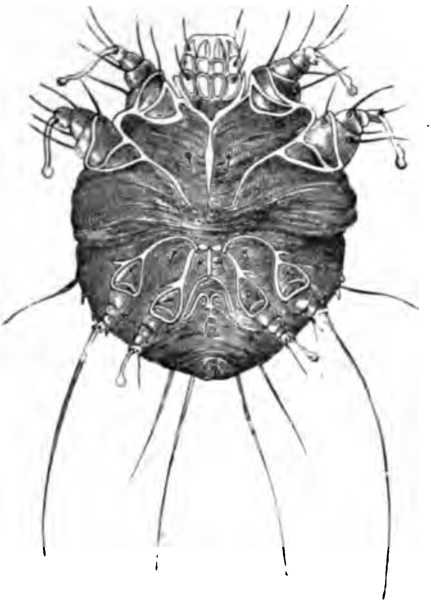
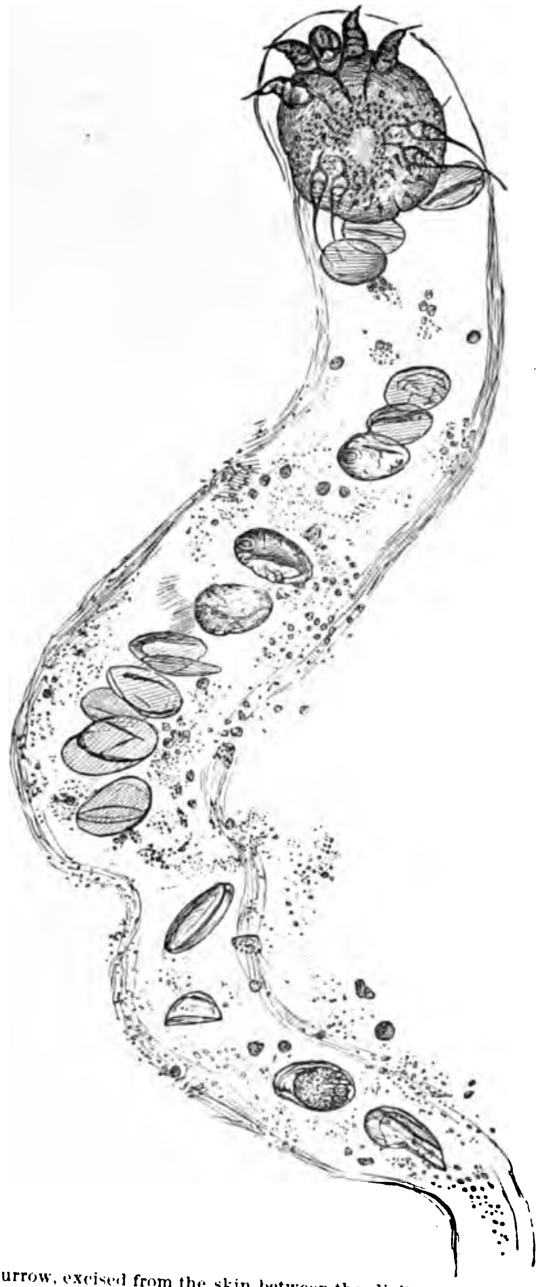


FIG. 38.—Male itch-mite, viewed from the ventral aspect; magnified 300 times (after Kaposi).



. 39.—Itch-burrow, excised from the skin between the digits; magnified 25 ti
sonal observation, Zurich clinic).

such grooves. The itch-mite especially prefers to make its groove in thin and comparatively juicy epidermis, and the burrows are found most frequently and in greatest number between the fingers, upon the flexor aspect of the wrist, in the axillary region, on the nipple, at the umbilicus, on the genitalia, over the trochanters, in the popliteal spaces, and in the gluteal region, as well as on the inner border of the foot. The face generally remains free, although in infants it is often involved if infection is acquired by application to the breast.

Papules, vesicles, and even pustules generally develop in the immediate vicinity of the grooves. The intolerable itching to which the itch-mites give rise, particularly from the heat of the bed, induces scratching on the part of the patient, and in this way a *secondary eczema* results, consisting of papules, vesicles, pustules, and crusts. Should crusts become superimposed upon one another, particularly upon the face and upon the scalp, and should, in addition, the nails become thickened and cracked, the condition has been designated *Norwegian scabies;* but this occurs only in neglected and long-standing cases. Enlargement of adjacent lymphatic glands—*buboes*—occurs as a *complication*, and occasionally suppuration may even take place. If patients with scabies are attacked by a disease attended with high fever, the itching of the skin ceases during the persistence of the fever, because the female parent dies as a result of the elevated temperature, but the itching recurs after defervescence as soon as new acari have developed from the ova.

Prognosis.—The prognosis of scabies is good, for the disorder can be cured with certainty within a short time.

Treatment.—Among the **prophylactic measures** frequent change of body-linen and bed-linen and treatment of mange in animals may be mentioned. The simplest remedy for the cure of scabies consists in a combination of **balsam of copaiba and liquid styrax**, with which the skin should be rubbed morning and evening for two days in succession, in order to destroy the **acari** and their ova:

℞ Balsam of copaiba,
  Liquid styrax, 50.0 (1½ fluidounces).—M.
For inunction twice daily.

On the third day the patient should take a **hot bath**, and wash the skin with green soap. At the same time clean body-linen and clothing are put on, and the bed-linen is changed. The soiled linen should be thoroughly boiled and washed. The clothing should, if possible, be disinfected by exposure to the action of live steam, thus in a sterilizing apparatus. Often the eczema resulting from the scabies will require treatment in the usual manner for some time.

Among many other *remedies for scabies* it will suffice to mention further *inunctions of petroleum, sulphur, lime, tar,* or *soap.* In hospitals the *solution* of *Vlemingkx* is much employed:

℞ Lime,	25.0 (6 drams);
Sulphur,	50.0 (1½ ounces);
Digest with rain-water,	500.0 (1 pint), and
Evaporate to	300.0 (9½ fluidounces).—M.
For inunction.	

Excessive friction of the skin should be avoided, as albuminuria and acute nephritis may result therefrom.

ACARUS FOLLICULORUM.

The acarus of the hair-follicles is a harmless parasite for human beings, finding lodgment within the sebaceous follicles, as many as twenty being present in a single follicle. It is especially common upon the bald head in the aged. If sebaceous matter be expressed with the finger-nails, and be smeared upon a slide with a drop of oil, the parasite can be readily recognized from its slender form and its four pair of feet (Fig. 40).

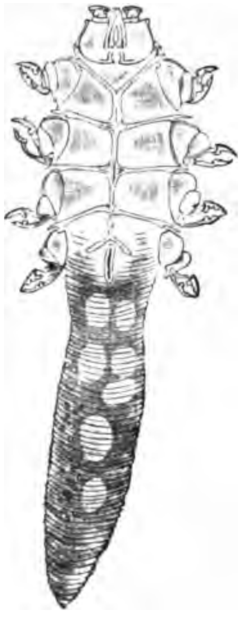


Fig. 40.—Acarus folliculorum (after Landois).

PEDICULI (LICE).

Three varieties of pediculi occur in human beings, namely, the *head-louse—Pediculus capitis,* the *crab-louse—pediculus* or *Phthirius pubis,* and the *clothing-louse—Pediculus vestimentorum.* Among these the clothing-louse is the largest, the crab-louse the smallest.

HEAD-LOUSE (PEDICULUS CAPITIS).

Head-lice are encountered only upon the scalp. The females, which always exceed the males in number, and are larger than the latter, attain a length of 2 mm. With their curved, six-hooked feet (p. 218, Figs. 42, 43) the lice are well able to climb upon the hairs. The fertilized female in this process deposits its ova or *nits* upon the hair, and these are readily recognizable as gray dots. On microscopic examination, the ova are found to be attached to the hairs by means of a chitinous sheath (p. 217, Fig. 41). The fertility of the pediculi is remarkable, as a single female parasite may deposit as many as 5000 ova within a period of six weeks. Within from three to eight days the y ova, and these will have b

Pediculi cause itching velops in consequence of and crusts, which usually appear in greatest abundance upon the occiput, which is the principal seat of the itching. Often the adjacent postcervical lymphatic glands are enlarged—*consensual bubo*—and these occasionally undergo suppuration and rupture externally. Often the hair is matted together by pus, blood, and crusts into an entangled meshwork with a disagreeable odor, on extrication of which innumerable ova and pediculi come into view. In marked cases the condition has been designated *plica polonica*, because it has been observed in its most developed form in the filthy inhabitants of certain parts of Poland. It was formerly believed to be dangerous to remove plica polonica.

Sleep is often disturbed in patients with pediculi on account of the itching, and they become pale and nervous and greatly exhausted.

Pediculi are acquired by contagion, as, for instance, in railway-journeys, from bed-companion infrequent combing of the may, therefore, occur oft in women who have pass tinued illness, and whose hair has not been carefully and regularly combed for a long time. For the recognition of pediculosis of the head the demonstration of the ova is important in addition to that of the parasites themselves. Pediculi can be destroyed by inunctions of *mercurial ointment*, which therefore has also been designated pediculi-salve. For the removal of the ova the hair must be disentangled and be washed with *vinegar*. The most rapid and the most certain remedy would consist in cutting the hair short. The *eczema* due to scratching should then be treated

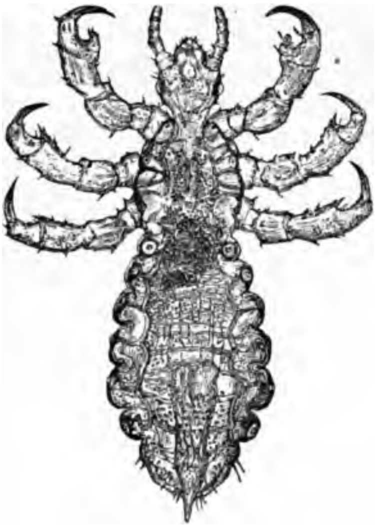


FIG. 42.—Male head-louse; magnified 25 times (from personal preparations).

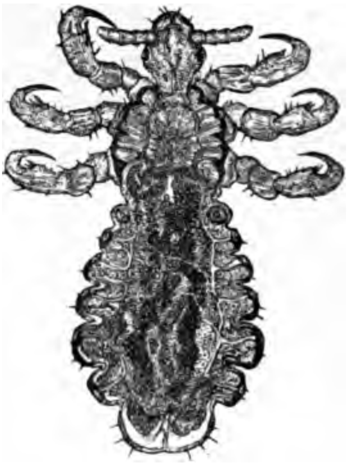


FIG. 43.—Female head-louse filled with blood; magnified 25 times.

CLOTHING-LOUSE (PEDICULUS VESTIMENTORUM).

The clothing-louse, which attains a length of from 3 to 5 mm., is found especially between the folds of the shirt. In this situation it deposits its ova in a garland-like arrangement, and from this point it reaches the skin and bores its way with its proboscis to obtain blood. On removal of the clothing the pediculi are not found upon the skin, but must be looked for between the folds of the shirt. They occur in especially large number upon the neck, between the scapulæ, in the gluteal region, over the sacrum, on the outer aspect of the thigh, and at the wrist-joint. The skin is in part covered with bite-marks, and in part it presents papules, pustules, and crusts. The latter are the result of scratching, as clothing-pediculi cause intense itching.

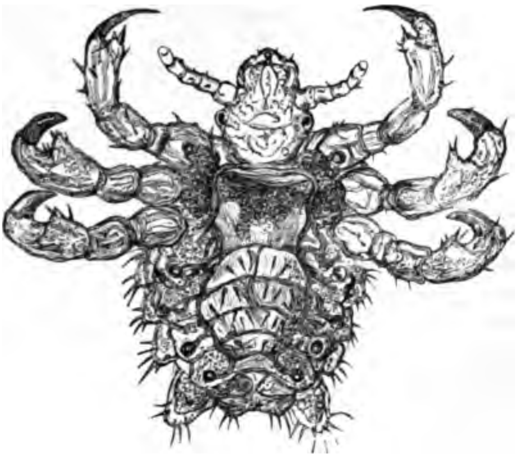


FIG. 44.—Crab-louse; magnified 25 times (from personal preparations).

Clothing-pediculi are found especially in vagrants, who mutually infect one another in sleeping together and through other intimate association. Railway-journeys, sea-voyages, as well as clothing-establishments, may be the means through which infection of respectable individuals takes place. When clothing-pediculi have been present for a long time the skin gradually acquires a brownish, even a blackish, color—*nigrities*—suggestive of Addison's disease, and resulting from transformation of the hemoglobin of the extravasated red blood-corpuscles. For the removal of clothing-pediculi the clothing should be boiled and be thoroughly cleansed with soap, and then be placed in a room at a temperature of 80° C. (176° F.), in order to destroy the pediculi, and the usual treatment should be employed for the relief of the eczema due to scratching.

FLEAS (PULICES).

The *common flea—Pulex irritans*—lodges between the folds of the body-linen, whence it attacks the skin, in which it makes a puncture and then sucks blood. The *puncture* can be readily recognized from its bloody appearance, and is surrounded by a *roseola* due to cutaneous hyperemia. The latter, however, soon disappears, so that only the site of the puncture persists for some time. In persons with a delicate skin, particularly children, *wheals* also form. In persons of unclean habits the skin often is covered with innumerable flea-bites. Capture of the fleas and the use of insect-powder are certain means for their extermination.

The *sand-flea—Pulex penetrans*—occurs in America. The female bores deeply into the skin in order to fill itself with blood, and causes, as a result, inflammation and destruction of the skin, which may lead to erysipelas, lymphangitis, gangrene of the skin, and even tetanus.

BEDBUG (CIMEX LECTULARIUS).

The bite of the bedbug is followed by the development of *wheals* upon the skin and *itching*. Occasionally generalized *urticaria* results. Often various *scratch-efflorescences* are encountered. Thorough cleansing of the bedsteads and of the bed-linen and the body-linen will suffice for the destruction of the parasites.

CYSTICERCUS OF THE SUBCUTANEOUS CONNECTIVE TISSUE.

Cutaneous cysticerci appear in the form of peculiarly hard and generally flattened nodules up to the size of a cherry, over which the skin presents an unchanged appearance. They are readily movable upon the subjacent tissues, and can be readily mistaken for enlarged lymphatic glands. A positive diagnosis can be made only by excising a nodule with the knife and finding a dense-walled vesicle filled with fluid and exhibiting a slight depression at one point, where are situated the neck and the scolex of the tapeworm often in movement. Occasionally only a few vesicles are present, while in other instances their number may reach hundreds. Here and there individual cysticerci become smaller and finally disappear, while in other situations new cysts appear. Occasionally the same individual may present also cysticerci in the eye or the brain, and he will, accordingly, complain of visual disturbance or of epileptic convulsions, although he will but rarely be the host of a tapeworm at the same time. In the latter event auto-infection would be possible only by the migration of segments of tapeworm (proglottides) into the stomach, where they are digested, so that the ova are set free. No internal remedy is known that is capable of destroying cysticerci of the skin, so that the only remedy is removal with the knife, and this will be possible only when a small number of cysts are present.

Among the parasites of the skin the *whip-worm—Filaria medinensis*—may further be mentioned. This parasite occurs upon the west coast of Africa, attains a length of one meter, and bores its way deeply into the skin, where it gives rise to destruction, furuncles, and even gangrene. It should be noted whether the worm can be seen to protrude from a wound in the skin, and then it should be slowly removed by winding it upon a bit of wood.

The ***mosquito*** (*Culex pipiens*), the *dog-tick* (*Ixodes ricinus*), the ***harvest-mite*** (***Leptus autumnalis***), the *barley-mite* (*Acarus hordei*), and the ***bird-mite*** (***Dermanissus avium***) may also be mentioned as occasional parasites of the skin.

VEGETABLE PARASITES OF THE SKIN (DERMATOMYCOSES).

PITYRIASIS VERSICOLOR.

Pityriasis versicolor results from the invasion and the proliferation of a *mold*, the *Microsporon furfur*, between the horny cells of the epidermis, and is attended with the presence of *brownish* ***spots*** upon the skin, which are somewhat raised above the level of the adjacent healthy skin, are not glistening, and readily undergo desquamation when rubbed with a hard body, such as the finger-nail. If the scales are cleared up with potassium hydroxid upon a slide, the round conidia and septate and ramifying myce-

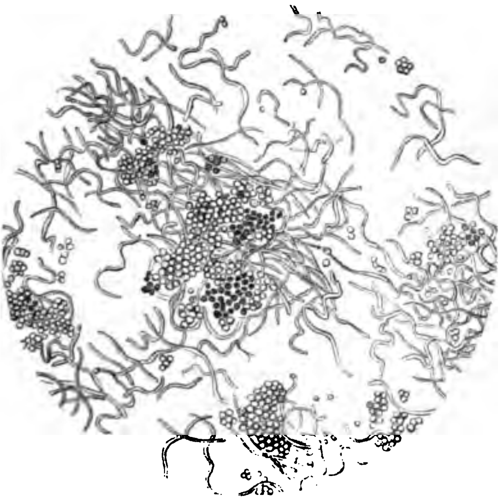


FIG. 45.—Microsporon furfur: mycelial filaments and conidia: potassium-hydroxid preparation; magnified 250 times (personal observation, Zurich clinic).

lial filaments of the fungus come clearly into view beneath the microscope, and render the diagnosis certain (p. 221, Fig. 45). The *spots* are at first small and barely visible, but become larger and coalesce, and finally large areas of skin are involved. Often the skin presents a yellowish and brownish spotted appearance. The changes in the skin appear earliest and most constantly upon the *covered parts of the body*, because in these situations the skin is moist and warm, and the proliferation of the fungus is thereby favored. The skin of the chest generally presents the first alterations, and then the abdomen, the neck, and the back, and, finally, also the genitalia and the adjacent surfaces of the thighs are involved. The face almost always escapes, and the hands and the feet always. Only rarely does inoculation of one person by another take place, as, for instance, between occupants of the same bed or between husband and wife. Children and old persons are generally not attacked, so that individuals with pityriasis usually are relieved spontaneously of their disorder in old age. Individuals with a delicate skin, and with a tendency to sweating, therefore also patients with pulmonary tuberculosis, are attacked by pityriasis versicolor with especial frequency. The disorder is *unattended with danger* and is *curable*, and at most gives rise to itching from the sweating, and is principally annoying to the patient on account of the disfigurement, which naturally only becomes apparent on undressing, as, for instance, in bathing. The fungi can be removed mechanically by *inunction of the skin with green soap* at night, followed by a *bath* the next morning; or the fungi may be destroyed by painting the skin with *oil of turpentine*. The following formula may be recommended:

℞ Lactic acid,
Salicylic acid, each, 5.0 (75 grains);
Elastic collodion, 20.0 (5 fluidrams).—M.

The discolored areas of skin are painted with this solution, which is applied also for a considerable distance beyond, and the detachment of the coating of collodion is awaited. It is important in connection with every form of treatment not to overlook any affected portion of skin, as recurrence might speedily take place from such an area.

FAVUS.

Etiology.—Favus results from the proliferation in the *hair-follicles* of a *filamentous fungus* known as the *achorion* or *Oïdium Schœnleinii*. In the majority of cases the scalp is affected, and only rarely are other portions of the body covered by hair invaded.

The well-known clinician of Zurich, and subsequently of Berlin, Schœnlein, discovered in 1839 the mold named in his honor. Recently several (as many as five) varieties of favus-fungi have been distinguished, and different pathogenic properties attributed to each, although it is by many believed, upon good evidence, that there is really but a single favus-fungus, and that the apparent variations are dependent upon minor accidental conditions, such as the character of the nutritive medium.

Favus is a rare disease at the present day, because cleanliness in the care of the scalp is no longer the privilege of those in better circumstances, but has become a necessary condition of life even among the poorer classes. Inoculation may take place from one individual to another, or from animals to human beings. For **inoculation of one person by another** to take place, intimate and prolonged association with a patient suffering from favus is necessary. This generally occurs among those who *sleep together*. In impoverished and filthy families favus occasionally occurs as a *familial disease*, one member of the family inoculating another. Among animals, favus occurs not rarely in *mice* and *rats*, and from these the affection is readily conveyed to cats. The disorder, however, is encountered also in dogs, cows, rabbits, hens, and ducks. **Association with animals affected with favus** is sufficient for the inoculation of human beings. Favus is generally observed in *children*, and in persons *under the age of twenty-five years*. *Seborrhea of the scalp* appears to favor lodgment of the fungi. Experience has shown that *males* are attacked more commonly than females.

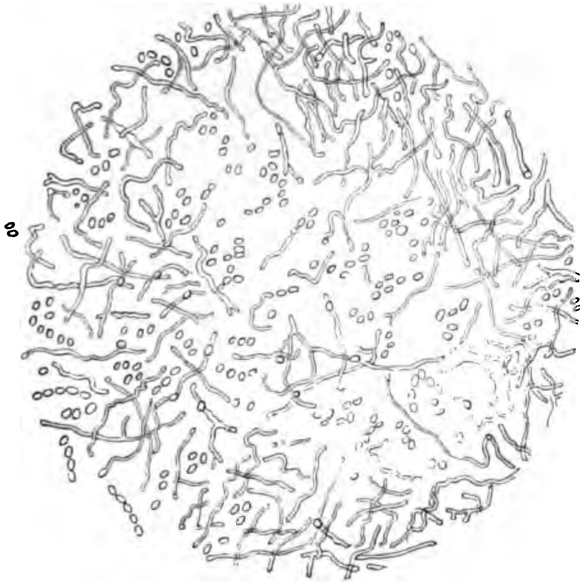


FIG. 46.—Achorion Schœnleinii, from a case of favus: magnified 275 times (personal observation, Zurich clinic).

Symptoms and Diagnosis.—The earliest changes upon the scalp consist in the appearance of yellowish spots in the hair-follicles that progressively increase in size and project from the adjacent skin in the form of sulphur-yellow, dry, crust-like deposits. The latter are known as *favus-bodies*, and generally possess a dish-like shape suggestive of crabs' eyes, so that the condition has been designated *scutellate* or *urceolar favus.* If favus-bodies be rubbed up, they give off a distinctive moldy odor, suggestive of that of the urine of mice, and if they be touched with alcohol the yellow color becomes more intense. On teasing small portions in potassium hydroxid they become clear, and on microscopic examination they will be found to consist of conidia and mycelial filaments (p. 223, Fig. 46). The conidia are situated especially in the deep layers, and exhibit an oval and cylindrical shape, while the mycelial filaments are more numerous in the upper layers and appear as septate, and frequently divided, small filaments. The nutrition of the *hairs* is soon disturbed by the favus-fungi that have entered their follicles. The hairs become dry, appear as if covered with dust, and readily break. If the hair be extracted, cleared in potassium hydroxid, and examined microscopically, conidia and mycelial filaments will be found upon them, and the diagnosis is thus rendered certain (Fig. 47).

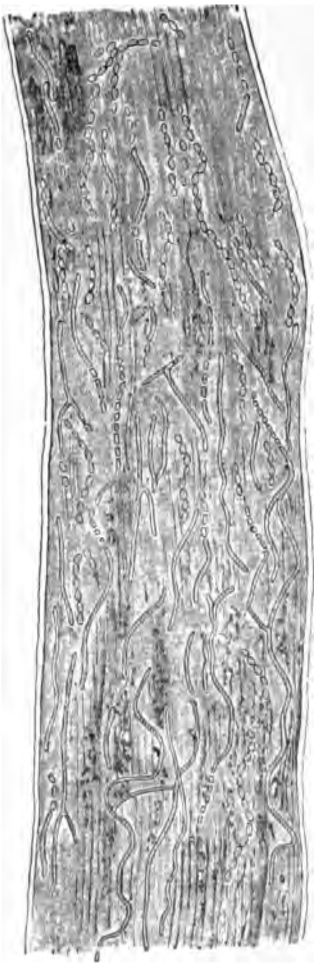


FIG. 47.—Hair from a case of favus, showing conidia and mycelia: magnified 275 times (personal observation, Zurich clinic).

Careful examination shows that the favus-fungus first becomes attached in the funnel-like orifice of a hair-follicle, and then proliferates downward between the root-sheaths of the hair. Gradually it bores its way through the superficial coat of the hair and then penetrates the hair-structure proper.

From what has been said it can be readily understood that within the area of the favus-crusts only stumps of hairs are found, or no hair at all. Adjacent favus-crusts coalesce, and often cover the scalp throughout a large extent, and this condition has been designated *favus confertus*. The baldness to which favus gives rise is curable only if the favus-fungi have not involved the papillæ of the hair, and it remains permanent if the hair-papillæ are destroyed. If in cases of favus of long standing the crusts are detached from the skin, the subjacent tissues often appear reddened and thinned. Occasionally atrophy of the bones of the skull from pressure is said to have taken place. Sometimes the skin is the seat of eczema, which at times extends beyond the limits of the hair to the adjacent skin. The patients exhibit no symptoms beyond **itching**, and if sufficiently indifferent they may bear their disease occasionally for more than thirty years. Often isolated crusts are thrown off spontaneously, but perfect recovery does not take place without the employment of medicinal measures. Some patients present, further, *favus of the nails*—*onychomycosis favosa*—if in scratching the scalp the nails have become inoculated. The nails then appear thickened, readily peel, and if portions of nail are cleared up in potassium hydroxid and examined microscopically, favus-fungi will be found. Favus occurs but rarely in other situations, but always only on parts of the body **covered with hair.** In such situations individual crusts more readily fall off. Favus-fungi have been found also in necrotic areas of the gastro-intestinal mucous membrane in a case of favus.

Prognosis.—Favus is unattended with danger to life and is susceptible of cure, although at times baldness remains permanent.

Treatment.—Among the **prophylactic measures** the extermination of the favus of animals and care in association with individuals who have favus should be mentioned. In the treatment of a case of favus all crusts must first be removed by means of **applications of oil.** With this object in view the crusts should be painted every two hours with olive-oil and be covered with cotton or flannel saturated with olive-oil. Only after the crusts have been removed should all of the diseased hairs be gradually extracted by means of **ciliary forceps**, in order to remove the fungi from the skin with them. In addition, the skin should be painted freely morning and evening with **oil of turpentine** in order to destroy the fungi. The treatment must be persisted in for many weeks, as it may readily happen that a few diseased areas of skin remain concealed or are not accessible, and from these renewed extension of the disease may take place. In cases of *onychomycosis favosa* the diseased portions of the nails are removed with **scissors,** the incision extending beyond the diseased area, and the margin of the incision is painted with mercuric-chlorid collodion (1 : 20).

HERPES TONSURANS.

Etiology.—Herpes tonsurans is dependent upon the invasion of a **mold,** the *Trichophyton tonsurans,* which at times proliferates upon the scalp, but at other times also upon other portions of the skin. **Inoculation of one person by another** occurs more frequently than in the case of favus, because the trichophyton adheres more readily to the human skin. Epidemics of herpes tonsurans have, therefore, often been observed in educational institutions, orphan-asylums, and barracks. Not rarely epidemics have originated from *barber-shops,* inoculation taking place through inadequately cleansed tonsorial instruments that had previously been employed upon a patient suffering from herpes tonsurans. **Inoculation of human beings by animals** may also take place, as herpes tonsurans is not at all rare in dogs, cats, horses, calves, cows, and rabbits. Herpes tonsurans not rarely arises spontaneously in **damp dwellings,** after the application of **poultices** and **affusions of hot water,** and after the employment of **cold-water procedures,** obviously because proliferation of the fungus is thereby favored. In general, herpes tonsurans occurs more frequently in *damp weather,* as, for instance, in the spring, than during the dry periods of the year. *Children* and *young persons* are attacked with especial frequency. The disorder occurs not rarely, also, among the *better classes.*

Symptoms and Diagnosis.—It is customary to distinguish three varieties of herpes tonsurans, and to designate them macular, vesicular, and squamous. *Macular herpes tonsurans* is attended with the presence of a pale-red or brownish-red spot in the affected region, around which, after a time, a new circle of reddened skin appears. Gradually new circles constantly develop, and in this way the abnormal condition of the skin extends progressively. The central portions meanwhile have not rarely acquired a normal appearance again, while the peripheral changes gradually extend. The redness of the skin is generally followed by the formation of scales, and macular herpes tonsurans is then transformed into squamous herpes tonsurans. *Vesicular herpes tonsurans* is attended with the formation of small vesicles upon the skin, which, after a time, undergo desiccation, and then form scales rather than crusts. Around the vesicles circles of new vesicles appear, and thus the process extends in exactly the same manner as the growth described for macular herpes tonsurans. In cases of vesicular herpes tonsurans also the central portions have not rarely become normal again, while the disease continues to progress at the peripheral limits. As the formation of vesicles is everywhere followed by desquamation, vesicular herpes tonsurans becomes gradually transformed into squamous herpes tonsurans. *Squamous herpes tonsurans* never occurs as a primary disease of the skin, but is always the result of an antecedent macular or vesicular herpes tonsurans.

In accordance with the *situation* in which herpes tonsurans appears, *herpes tonsurans of the scalp* may in the first place be distinguished. The trichophyton-fungus, like the favus-fungus, invades the hair-follicles, penetrating at first between the hair-sheaths, then, after destroying the superficial covering of the hair, into the substance of the hair itself, and causes changes in the latter. The hairs appear dry, and, in contradistinction from favus, acquire a chalky-white color on addition of chloroform, to the extent that fungi have invaded the hair. They are prone readily to yield to traction and to break, and there then result circular areas of skin covered with stumps of hair, which resemble a badly cared for tonsure, and from which the name of the disorder has been derived. In contradistinction from favus, the trichophyton-fungus does not penetrate so deeply into the hair-follicles, and it almost always leaves the hair-papillæ intact, and as a result the hairs grow again after removal of the fungus. Herpes tonsurans of the scalp generally occurs as the squamous variety, and it frequently extends beyond the actual boundaries of the hair, invading for a short distance also the adjacent nucha and the forehead.

Herpes tonsurans of the beard gives rise to the clinical picture of *parasitic sycosis*, which has been described on p. 182.

Herpes tonsurans of the skin can be readily recognized from its circular distribution. Naturally, the diagnosis is unequivocally certain only when the distinctive trichophyton-fungi can be demonstrated in the epidermic scales after previous clearing with potassium hydroxid. To this end the most peripheral portions must be examined, as the fungi frequently have already disappeared from the central portions. It also happens not rarely that fungi can nowhere be found, although in consequence of the characteristic appearance of the lesions in the skin there can be no doubt as to the nature of the disorder. In examining diseased hairs a large number must frequently be studied before the fungus is found.

Trichophyton-fungi, like favus-fungi, form roundish and cylindric conidia, and branched filamentous mycelia, filled with granules and vacuoles (Fig. 48), and it would not be possible to distinguish the fungi of favus and trichophytosis from the appearance of individual conidia and mycelial filaments. In the differentiation from favus the mycelial filaments predominate in the presence of herpes tonsurans, and they exhibit a lesser tendency to ramification. The fungi are situated in the skin between the horny and the mucous layer of the epidermis, and never penetrate the structures of the cutis. *Marginate eczema* represents a special variety of cutaneous herpes tonsurans. It is encountered upon the scrotum and the adjacent surfaces of the thighs, although it may extend also to the perineum and the sacral region. The skin appears greatly reddened and is often covered with vesicles, pustules, crusts, and scales. The sharp limitation from the healthy skin is especially distinctive. The demonstration of the trichophyton-fungus is again decisive in diagnosis. This peculiar variety of herpes tonsurans results from the application of adjacent portions of skin to each other and the consequent chafing and maceration. Marginate eczema therefore occurs also at other suitable portions of the body, thus at the umbilicus, beneath the fold of the breast, and the axillary cavity.

Trichophyton onychomycosis is attended with turbidity, thickening, fragility, and friability of the nail-tissue. The condition obviously results from auto-inoculation in scratching a diseased part of the skin. Occasionally recovery from the disease of the skin has already taken place, while the nail remains diseased. Herpes of the nails occurs, as experience has shown, more commonly than favus of the nails. The fungi can be readily demonstrated in the scales from the nails cleared up with potassium hydroxid.

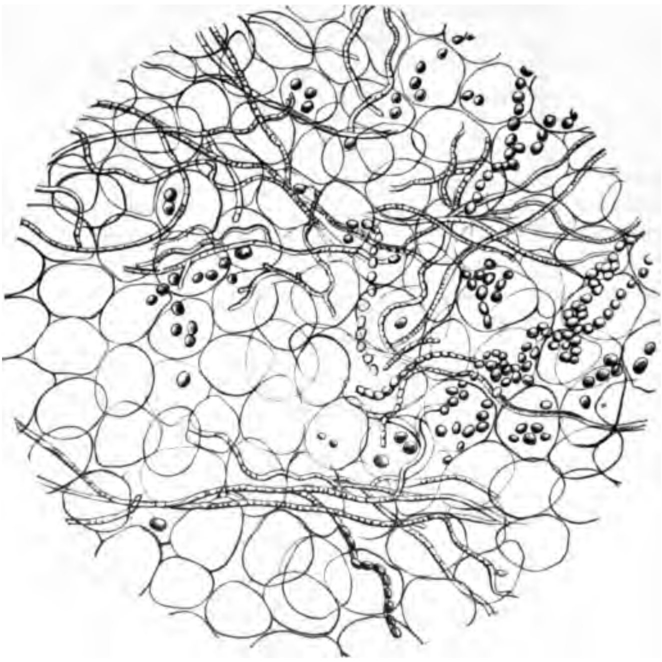


FIG. 48.—Epidermic scales from a case of squamous herpes tonsurans; potassium-hydroxid preparation; magnified 275 times (personal observation, Zurich clinic).

The *duration of herpes tonsurans* is occasionally protracted over many years, although *spontaneous recovery* may take place.

Prognosis.—The prognosis is favorable from every point of view. The disorder is never dangerous to life, and apart from itching it gives rise to no other symptom, and, besides, it is readily amenable to treatment.

Treatment.—The **prophylactic measures** consist in care in associating with individuals and animals suffering from herpes and in thorough cleansing of all tonsorial instruments. The treatment of herpes tonsurans is the same as that of favus (p. 225).

ERYTHRASMA.

Erythrasma occurs upon the same portions of the skin as marginate eczema, with which it is often confounded. It, likewise, is attended with the presence of reddish and brownish-red areas of skin, covered with crusts and scales, and characterized by their sharp margins. The fungus of erythrasma forms conidia and mycelial filaments, although the former are exceedingly small almost like micrococci, and the mycelial filaments also are conspicuous for their narrowness. The treatment is the same as that for pityriasis versicolor (p. 222).

PART VIII.

DISEASES OF THE SPLEEN AND THE BLOOD.

I. DISEASES OF THE SPLEEN.

EMBOLIC INFARCTION OF THE SPLEEN.

Etiology.—Embolic infarction of the spleen occurs most commonly in conjunction with **diseases of the mitral or aortic valves,** if thrombotic deposits upon the leaflets have been detached and have entered the arterial circulation, through which especially the distribution of the splenic artery is frequently reached by emboli. **Cardiac thrombosis, echinococcus of the heart, neoplasms of the heart, aortic aneurysms,** and **arteriosclerosis of the aorta** are far less frequently the sources for emboli of the spleen. An immediate exciting cause for the embolism is generally not demonstrable. By reason of the nature of the causative factors splenic emboli generally occur in adults.

Anatomic Alterations.—Emboli in the splenic artery do not, as a rule, remain lodged in the main trunk, but enter smaller arterial branches, in which they give rise to the formation of **wedge-shaped splenic infarcts.** The vascular area cut off from its arterial blood-supply becomes anemic, undergoes so-called coagulation-necrosis, and forms a wedge-shaped area at first rather pale gray, but subsequently yellowish in color, with its base directed toward and reaching the surface of the spleen, where it is readily recognizable, while the apex is directed toward the hilus of the spleen, and corresponds with the situation of the embolus. Only the boundaries of the wedge-shaped infarct are surrounded by a hyperemic zone a few millimeters wide. Gradually, **fatty degeneration** and **absorption** take place in the embolic vascular area, which becomes smaller and eventually contracts to a narrow cicatricial band, to which a depression upon the surface of the spleen corresponds. **Calcification** may also take place throughout the area of a wedge-shaped infarct of the spleen. Often *several infarcts* of the spleen are present together, having developed at different times, and accordingly presenting different appearances.

Symptoms and Diagnosis.—Embolism of the spleen is occasionally unattended with symptoms—*latent embolism of the spleen.* In other instances, however, the patient complains suddenly of **pain** in the splenic region, and, in addition, **acute enlargement of the spleen** is demonstrable, in connection with which it is of especial diagnostic significance that the spleen can be felt. It may also happen that splenic embolism is complicated by acute circumscribed *fibrinous perisplenitis* throughout the embolic area, and which may be recognized from the presence of a palpable or audible peritoneal friction-murmur. Occasionally a **chill, elevation of temperature,** and **vomiting** occur, manifestations that are generally considered as due to reflex irritation. The symptoms usually disappear in the course of a few days.

Prognosis.—The prognosis of infarction of the spleen is favorable so long as the embolus exerts only mechanical effects and is unattended with pyogenic bacteria. Should this not be the case, the infarct will be converted into a splenic abscess, which constitutes a dangerous disorder.

Treatment.—An **ice-bag** should be applied over the spleen in order to relieve the pain and to antagonize any possible inflammatory process that may have developed. In the presence of extremely severe pain **subcutaneous injections of morphin** will be necessary.

ABSCESS OF THE SPLEEN.

Etiology.—Abscess of the spleen scarcely occurs except through the activity of **pyogenic bacteria,** but up to the present little is known concerning the nature of these. Abscess of the spleen occurs with relative frequency in the sequence of **infectious diseases,** either the specific bacteria of the acute infectious diseases, or, as probably occurs more frequently, pyogenic cocci, particularly the Streptococcus pyogenes, giving rise to suppuration of the spleen. Abscess of the spleen is observed with especial frequency in connection with relapsing fever. *Embolic abscess of the spleen* occurs in association with *ulcerative endocarditis* if bacterial emboli gain entrance into the distribution of the splenic artery. *Traumatic abscess of the spleen* is the result of injury in the splenic region, induced by falls, shocks, contusions, or blows, and also by stab-wounds and gunshot-wounds. It is noteworthy that an abscess may form even when no external wound has been inflicted. Under such circumstances it must be assumed that the traumatism has lowered the resistance of the splenic tissue and thus rendered it more readily accessible to the invasion of pyogenic bacteria already circulating in the blood. Occasionally abscess of the spleen results

by extension from adjacent disease. Thus, it may occur as a complication of toxic gastritis, ulceration of the stomach, peritonitis, perinephritis, and gangrene of the lung. In some cases it is impossible to elicit a cause for the disorder, and under such conditions the designation *cryptogenetic abscess of the spleen* is employed.

Anatomic Alterations.—An abscess of the spleen may occasionally attain such proportions that no splenic tissue whatever remains, and the greatly distended capsule of the spleen contains more than five liters of pus. When abscesses of the spleen are of embolic origin several collections of pus are not rarely present. Under such conditions the abscesses may be encapsulated by connective-tissue membrane, or they may be free; in the latter event the wall appears ragged, flocculent, and often torn. The pus at times resembles ordinary greenish, creamy pus, while at other times it has acquired a reddish or brownish, chocolate-like appearance, in consequence of the presence of hemoglobin, and on microscopic examination it is found to contain polynuclear round cells, fatty granular cells, granular and fatty detritus, red blood-corpuscles, and crystalline hemoglobin.

Symptoms and Diagnosis.—In some cases abscess of the spleen is unattended with symptoms, and is discovered accidentally on post-mortem examination—*latent abscess of the spleen.* In other instances the clinical picture of **septicopyemia** (repeated chills, hectic fever, sweats, progressive emaciation, and pallor) is present, and a concealed focus of suppuration may be suspected, but whose seat is discovered only upon post-mortem examination. In a third group of cases **enlargement of the spleen, pain over the spleen,** and **septicopyemia** are present, but that these are dependent upon abscess of the spleen often becomes clear only on *sudden rupture of the pus.* Should this take place free into the peritoneal cavity, rapidly fatal peritonitis usually follows, although at times antecedent peritonitic adhesions prevent free rupture, so that an encapsulated accumulation of pus forms. Rupture takes place occasionally also into the stomach, the intestines, or the urinary passages, and under such circumstances there may occur, together with diminution in the enlargement of the spleen, vomiting of pus, or the appearance of pus in the stools or in the urine. Occasionally the pus ruptures through the diaphragm into the pleural cavity, the bronchi, or the pericardium, with the development of pyothorax, purulent expectoration, or pyopericardium respectively. Rupture of the pus externally also may occur, in connection with which it should be observed that the pus occasionally burrows for considerable distances beneath the skin, and possibly makes its appearance in the axillary cavity or beneath the left clavicle. The diagnosis becomes most positive if a **point of fluctuation** can be demonstrated in an enlarged and painful spleen; for, although echinococcus and soft carcinoma of the spleen likewise yield fluctuation, the presence of fever and the etiologic factors are in favor of abscess of the spleen. Spontaneous recovery from abscess of the spleen in consequence of *inspissation, absorption*, and *calcification of the pus* can be expected only in the presence of small abscesses.

Prognosis and Treatment.—The *prognosis* of abscess of the spleen is always serious, as in consequence of septicopyemia and rupture the danger is considerable. Internal remedies are incapable of effecting a cure, so that the only hope of success is afforded by **incision of the abscess with the knife.**

PERISPLENITIS (INFLAMMATION OF THE CAPSULE OF THE SPLEEN).

Etiology.—Inflammation of the capsule of the spleen occurs occasionally as a result of antecedent **traumatism.** It often attends **inflammatory processes in the spleen,** as, for instance, embolism or **abscess.** Not rarely it is one of the phenomena of **peritonitis.**

Anatomic Alterations.—A distinction should be made between acute and chronic perisplenitis. In the presence of *acute perisplenitis* the capsule of the spleen is covered with fibrinous deposits. These have often **formed pockets filled with pus,** in consequence of adhesions to adjacent organs. *Chronic perisplenitis* is attended with thickening of the capsule of the spleen. The thickened portions present a tendinous, white, and turbid appearance, and occasionally cut with cartilage-like hardness. Often the thickening is the seat of cicatricial contraction, so that the **surface of the spleen** is uneven. Under such circumstances the splenic tissue may have undergone considerable **atrophy from compression.** Frequently **adhesions** to adjacent viscera have formed, from which the spleen can be separated only with great difficulty. Not rarely the surface of the spleen is covered with numerous **villous appendages.**

Symptoms.—*Acute perisplenitis* is by no means always recognizable during life. In addition to **pain in the splenic region,** the presence of a **perisplenitic friction-murmur** is of especial diagnostic significance, and this may be appreciable on palpation and auscultation with the respiratory displacement of the spleen, occasionally, however, only on displacement of the abdominal walls upon the surface of the spleen with the hands. *Chronic splenitis* also may be attended with **pain in the splenic region** and **friction-murmurs.** Not rarely the **surface of the spleen** appears uneven to the **palpating** finger, and diagnostic difficulty may arise in consequence. If extensive adhesions are present, **respiratory displacement of the spleen** will be wanting. Enlargement of the spleen may further not occur under conditions that give rise to a splenic tumor (acute infectious diseases), and, finally, in cases in which the splenic percussion-dulness usually disappears (free perforative peritonitis) this may persist.

Prognosis.—The prognosis of perisplenitis depends upon the *causative factors.* Chronic perisplenitis is generally an insignificant disorder.

Treatment.—The treatment is purely symptomatic. Severe pain will require the application of an **ice-bag,** and, if necessary, **subcutaneous injection of morphin.**

AMYLOID SPLEEN.

Etiology.—Amyloid spleen, like amyloid degeneration in other organs, develops in the course of **chronic debilitating diseases,** generally attended with **wasting discharges,** such as suppuration of the bones and joints, chronic suppuration generally, malarial and syphilitic cachexia. Only rarely does it appear to develop without appreciable cause. Amyloid degeneration generally commences in the spleen, and then extends to the adrenal bodies, the kidneys, the liver, and the intestines. In the presence of suppuration three or four months are sufficient to bring about amyloid degeneration.

Anatomic Alterations.—In accordance with the appearances presented two varieties of amyloid spleen are distinguished —the *sago-spleen* and the *diffuse amyloid spleen.* In the *sago-spleen* the follicles appear upon the cut surface as gray, translucent granules resembling boiled sago, as the amyloid degeneration is principally confined to the follicles of the spleen. In the middle of the follicles the blood-vessels can often be recognized as dull-gray dots or lines. If tincture of iodin or Lugol's solution is poured over the cut surface of the spleen, the degenerated follicles assume a mahogany-brown color. The *diffuse amyloid spleen* is known also as the *ham-spleen,* because its cut surface presents a meat-red color, suggestive of smoked ham. In the presence of extensive degeneration the spleen is occasionally so much increased in size as to occupy a large portion of the abdominal cavity. It is generally hard, as if frozen, and its margin is blunt. The increased hardness is appreciable also on section. The splenic tissue is almost friable and translucent at the margins of the cut section. Tincture of iodin or Lugol's solution imparts a uniformly mahogany-brown color to the cut surface.

Commencing amyloid degeneration of the spleen can be recognized only by microchemic means, with the aid of a solution of iodin or methyl-violet (Vol. I., pp. 343, 344). The disease begins in the venous capillary spaces, and extends to the connective-tissue trabecula. The splenic cells remain often, though not always, unaffected. I do not share the view that the sago-spleen represents the commencement of the disease, which is followed later by diffuse amyloid spleen, as the process often does not advance beyond the sago-spleen, even in cases of long standing.

Symptoms and Diagnosis.—Amyloid spleen is unattended with symptoms, except that in the presence of marked enlargement of the organ **pressure-symptoms** may arise. In the diagnosis the demonstration of a **large, hard spleen, with a smooth surface,** is important, occurring in the course of chronic debilitating disease, especially if a large, hard liver, albuminuria, and edema and diarrhea are indicative of simultaneous amyloid degeneration of the liver, the kidneys, and the intestines.

The **prognosis** and the **treatment** are the same as those for amyloid liver (Vol. I., p. 344).

CARCINOMA OF THE SPLEEN.

Carcinoma of the spleen is a rare disorder. It may be *primary,* or, as occurs more commonly, *secondary* (after carcinoma of the stomach). Occasionally it has been observed in *children.* Most frequently the new-growth is a juicy *medullary carcinoma.* Not rarely *pigmented carcinoma* has been observed. The new-growth either forms circumscribed nodules, or infiltrates the splenic tissue in a diffuse manner. In diagnosis the detection of a large spleen with nodules, together with carcinoma in another organ (stomach, liver), is important. The patient complains principally of pain, and succumbs rapidly. Recovery is possible solely through *splenectomy,* although this should be performed only in the presence of primary carcinoma of the spleen.

Among other malignant new-growths *sarcoma of the spleen* may be mentioned, which gives rise to the same symptoms as carcinoma of the spleen, and during life is indistinguishable from the latter. Fibromata, enchondromata, cysts, dermoid cysts, lymphangiomata, and cavernomata also have been found in the spleen, conditions that either were discovered accidentally on post-mortem examination, or during life attracted attention in consequence of the enlarged and irregular spleen, and in a number of instances have been successfully removed by *splenectomy.*

ECHINOCOCCUS OF THE SPLEEN.

Either echinococcus occurs in the spleen exclusively, or it develops in conjunction with echinococcus in other organs, most frequently with echinococcus of the liver. Under such conditions the spleen at times becomes greatly enlarged, exerts troublesome pressure upon the stomach, the intestines or the urinary passages, and causes vomiting, constipation, or obstruction to the discharge of urine; or the diaphragm, the lungs, and the heart become so greatly displaced upward that death from suffocation is threatened. In diagnosis the detection of an enlarged spleen, covered by fluctuating prominences, is important, but a sense of fluctuation may be wanting over an echinococcus-cyst. Occasionally exploratory puncture may establish the diagnosis, if the fluid obtained contains an abundance of sodium chlorid, but is free from albumin, or if echinococcus-scolices or echinococcus-hooklets are present. Confusion with abscess of the spleen may readily occur if the echinococcus-cyst has undergone suppuration. A fatal issue may then readily result from septicemia.

Treatment.—Cure can be effected only through **operative measures.** It is possible for destruction and contraction of the cyst to be brought about by **injections of mercuric chlorid** (1.0 : 1000).

RUPTURE OF THE SPLEEN.

Reference will here be omitted to *traumatic rupture of the spleen,* such as may take place when the organ is healthy, in consequence of falls, shocks, blows, or contusions. The rupture of the spleen to be considered is that which occasionally occurs, particularly in connection with *acute splenic enlargement* in the sequence of acute infectious diseases (typhoid fever, typhus fever, relapsing fever, malaria, miliary tuberculosis), either spontaneously or as a result of cough, expulsive efforts, or other violent physical exertion. With the occurrence of the accident, the patient generally cries aloud with sudden *pain in the region of the spleen,* frequently makes the statement that something within the abdomen has torn, fails rapidly, and exhibits the *manifestations of internal hemorrhage* (increasing pallor, small, scarcely palpable pulse, feeble heart-sounds, obscuration of the field of vision, roaring in the ears, loss of consciousness). Occasionally death occurs rapidly amid chronic convulsions. In other instances the symptoms of circumscribed or generalized *peritonitis* appear. The area of splenic percussion-dulness is generally found increased. The complication is serious under all circumstances, and generally culminates fatally. In recent cases the abdomen is found filled with blood and softened splenic tissue.

The **treatment** consists in the administration of **stimulants** (subcutaneous injections of camphorated oil, internal administration of cognac or champagne), the application of an **ice-bag over the spleen, subcutaneous injections of extract of ergot,** and in the presence of severe pain **subcutaneous injections of morphin.** Recently, **celiotomy** and **splenectomy** have been successfully performed.

WANDERING SPLEEN.

The designation wandering spleen is applied to the condition in which the spleen has left its usual situation and has fallen to a greater or lesser depth in the abdominal cavity. Occasionally the organ may be found in one of the iliac fossæ, or even in the true pelvis. Generally a wandering spleen is at the same time a *movable spleen,* so that it can without difficulty be moved about and displaced in the abdominal cavity, and it also undergoes changes in position in accordance with the position of the body. Less commonly the spleen is attached in its abnormal situation, and it may perhaps have formed firm connective-tissue adhesions with the bladder or the intestines. The spleen is generally situated with its hilus directed upward, while its convex border is directed downward.

In **diagnosis,** the detection of depressions or notches in the border of the spleen in contact with the anterior abdominal wall is of special importance. In addition, consideration should be attached to the tongue-shaped form of the organ and the absence of splenic dulness in the usual situation. In isolated instances pulsation of the splenic artery could be felt in the hilus of the spleen. In many instances the organ is enlarged. Generally the viscus can be detected only by means of palpation; although in a woman with relaxed abdominal walls it was also visible. In many instances the condition is *latent,* and is discovered accidentally on careful examination of the abdominal viscera.

Symptoms.—Some patients complain of a **disagreeable sense of traction** and of **pain from pressure.** Occasionally **pressure-phenomena** referable to various abdominal viscera appear, as, for instance, difficulty in micturition, constipation, even intestinal obstruction, paresthesiæ, and weakness in one leg if the wandering spleen exerts pressure upon the nerves for the legs in the pelvis.

Etiology.—Wandering spleen is encountered much less commonly than wandering kidney, but it occurs more frequently in *men* than in women. **Traumatism** (falls, shocks, blows, constriction, expulsive effort, lifting) is often the principal exciting cause. The condition develops with especial readiness if the **spleen is enlarged.** It therefore occurs often in consequence of *malaria.* I have also observed a *leukemic wandering spleen.* The development of a wandering spleen is favored by **congenital relaxation** and **undue length of the gastro-splenic ligament.** At times wandering spleen is associated with wandering liver or wandering kidney.

Prognosis.—As a rule, the disorder is annoying rather than dangerous.

The **treatment** is purely mechanical. Naturally but little can be accomplished by means of **bandages. Splenectomy** and **splenopexy (suture of the spleen)** have been recommended, although such procedures will be resorted to only in the presence of severe symptoms or grave complications.

Permanent *displacement or dystopia of the spleen* may occur as a congenital condition in connection with *transposition of the viscera.* Less commonly the latter condition is confined to the liver and the spleen, so that the spleen occupies the right and the liver the left hypochondrium. Generally the remaining thoracic and abdominal viscera also are involved in the transposition.

ANEURYSM OF THE SPLENIC ARTERY.

Aneurysm of the splenic artery is a rare condition. Occasionally a *pulsating tumor in the abdominal cavity* has been demonstrable, while in other instances death has occurred unexpectedly amid symptoms of *internal hemorrhage*, the cause for which is found on post-mortem examination to be a ruptured aneurysm of the splenic artery. The aneurysm has also been known to form adhesions to the wall of the stomach, rupturing into this viscus, and giving rise to *fatal hematemesis*.

II. DISEASES OF THE BLOOD.

LEUKEMIA.

Etiology.—Leukemia consists in a *persistent and gradually progressive increase in the number of colorless corpuscles in the blood, together with a diminution in the number of red blood-corpuscles.* The disease is an *uncommon* one, which experience has shown generally avoids children and occurs *more frequently in men* than in women. Antecedent **infectious disease** and **traumatism** are alone known with certainty to be causative factors. Among the infectious diseases *malaria* and *syphilis* particularly should be mentioned, and among traumatisms injuries of bones or in the splenic region especially are deserving of consideration. To what extent *exposure to cold*, *anxiety*, and an *impoverished mode of life* operate as causes of leukemia is beyond definite determination. The disorder has frequently developed after **pregnancy** and the **puerperium.** I have observed leukemia to appear after a severe attack of **gout.** **Chronic diarrhea, rachitis,** and **scrofulosis** have further been mentioned as causes of leukemia. It is noteworthy that in some families leukemia occurs in *hereditary and familial distribution.*

Symptoms.—From the purely clinical point of view it is customary to distinguish *three varieties of leukemia*, and to designate them *lymphatic*, *lienal* or *splenic*, and *myelogenic* or *medullary.* These designations are employed accordingly as the lymphatic glands, the spleen, or the bone-marrow exhibits alterations, and it is accordingly probable that one or the other is to be looked upon as the principal seat for the generation of the increased number of colorless corpuscles in the blood. The blood-investigations of Ehrlich may, however, be fatal to this purely clinical view, and soon furnish the basis for a new classification of leukemia.

While it was formerly believed that lienal leukemia is the most frequent variety, and the myelogenic leukemia discovered by Neumann is the least common, it has recently been shown that the latter is actually the most frequent variety, and that formerly deception arose from the fact that pure varieties of leukemia are altogether rare, and that *mixed forms of leukemia* generally occur, in which disease of one blood-forming organ is soon complicated by secondary involvement of another, so that especially when the bone-marrow is affected its primary disease may be readily overlooked. Ehrlich attributes a subordinate significance to the spleen in the development of leukemia.

Of all of the symptoms of leukemia, the most important are the **changes in the blood and the blood-forming organs** (**lymphatic glands, spleen, bone-marrow**). Among the alterations in the blood, the **permanent increase in the number of colorless blood-corpuscles** is of distinctive significance. In diagnosis microscopic examination of a single small drop of blood obtained by means of a needle from the cleansed tip of the finger or lobule of the ear often is sufficient, for, while healthy blood generally contains from one to three colorless corpuscles in the field of the microscope, the number of colorless corpuscles (leukocytes) has occasionally undergone such enormous increase in cases of leukemia that it equals the number of red blood-corpuscles (erythrocytes), and in rare instances even exceeds this. It is noteworthy, even in fresh blood-preparations, that the character of the colorless corpuscles varies in accordance with the variety of leukemia. In cases of lymphatic leukemia the colorless corpuscles are generally smaller than the red corpuscles. They possess a large nucleus, which occupies almost the entire cell, and leaving only a small peripheral zone of protoplasm (so-called lymphocytes). In cases of lienal and myelogenic leukemia the majority of colorless blood-corpuscles are larger than the red. Cells with coarse, glistening granules are also frequently present (eosinophile cells). The occurrence of cells containing fatty granules and of nucleated red corpuscles (so-called transitional forms), which are thought to originate in the bone-marrow, is also of especial significance.

In the more exact study of the colorless blood-corpuscles the *color-analytical blood-examinations* devised by Ehrlich are indispensable, but these are applicable only to *dry preparations of the blood.* The latter may be made by spreading a thin layer of blood upon the surface of cover-slips, then adding a mixture of equal parts of alcohol and ether, which is permitted to evaporate in the air, or the cover-slips are dried and fixed upon a copper plate heated by a gas-flame. The dry preparations are then exposed to the action of solutions of certain aniline stains or mixtures of various aniline dyes, among which especially the *triacid solution* of Ehrlich and the *eosin-hematoxylin solution* of Ehrlich, may be recommended. By this means, colorless corpuscles with and without granulations can first be differentiated. The lymphocytes, for instance, contain no granulations. Among the colorless blood-corpuscles with granulation some can be recognized that stain with acid aniline dyes, as, for instance, eosin; they are, therefore, designated *acidophile* or *eosinophile cells.* It is especially these cells that are increased in number in leukemic blood when the bone-marrow is the seat of disease, and they are especially valuable in the diagnosis of beginning leukemia. Other colorless blood-corpuscles contain granules that can be stained by basic aniline dyes, such as methylene-blue—*basophile cells* or *mast-cells.* These are present in normal blood in extremely small number, while they are more numerous in leukemic blood, when the bone-marrow is the seat of disease, and they are almost even more significant in diagnosis than the eosinophile cells. The *neutrophile colorless blood-corpuscles* further should be mentioned, among which again those with several nuclei (*polynuclear*), those with a single nucleus (*mononuclear*), and those with a constricted or lobulated nucleus (*transitional forms*) are distinguishable. In triacid preparations the neutrophile leukocytes can be recognized from the fact that they acquire a violet tint. In cases of leukemia large mononuclear neutrophile leukocytes especially appear, which are derived from the bone-marrow and are designated *myelocytes*. These also are important in the diagnosis of leukemia, as they are not present in normal blood.

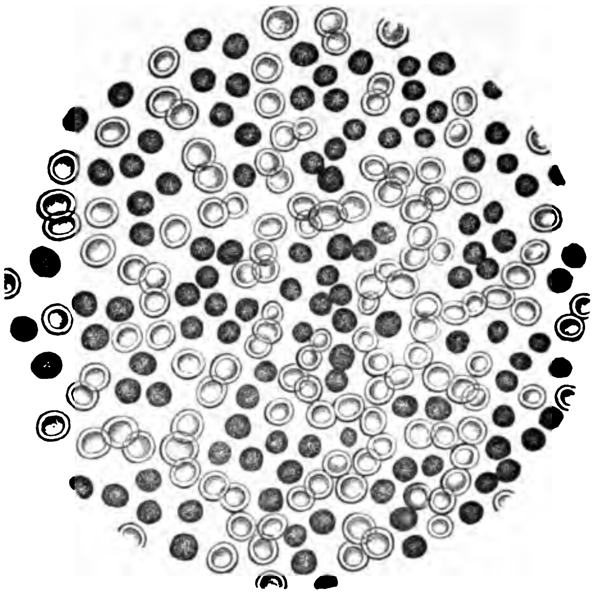


FIG. 49.—Blood from a case of predominantly lymphatic leukemia; magnified 450 times (personal observation).

In order to determine the *number of colorless corpuscles in the blood* the Thoma-Zeiss hemocytometer may be employed. In healthy individuals the blood contains from 6000 to 8000 colorless, and from 4,000,000 to 5,000,000 red blood-corpuscles in 1 c.c., so that the proportion of white to red would be about 1 to 500. If large amounts of blood are available, the increase in the number of colorless corpuscles may be demonstrated macroscopically by means of the *blood-sedimenting method* of Welker. If blood be introduced into a high glass cylinder and be permitted to stand quietly, a red sediment, consisting of red blood-corpuscles, falls to the bottom, over which a much smaller grayish-white layer of colorless blood-corpuscles forms, and over this a layer of blood-serum. In cases of leukemia the gray middle layer undergoes a considerable and readily recognizable increase, in correspondence with the increase in the number of colorless blood-corpuscles. It is noteworthy further that *ameboid movements of colorless blood-corpuscles* are but slight or are wholly wanting.

The *red corpuscles* in leukemic blood are diminished in progressively increasing degree, and in conformity therewith the *percentage of hemoglobin* also becomes reduced. For the determination of the latter the hemoglobinometer of Gowers may be employed.

Sometimes the red blood-corpuscles exhibit excessive *polymorphism*, *poikilocytosis*, and pear-shaped, figure-of-eight shaped, dumb-bell shaped cells and cells resembling the root of a tooth are observed. Such alterations occur in connection with all possible varieties of profound anemia. *Ameboid movement* and *constrictions of red blood-corpuscles* are also without significance and are related to the anemic state. For the demonstration of *nucleated red blood-corpuscles* or *transitional forms* the hematoxylin-eosin solution of Ehrlich should be employed. In dry preparations of the blood thus treated the red corpuscles acquire a bright-red hue, while such nuclei as are present will be stained deeply blue and occasionally exhibit mitoses.

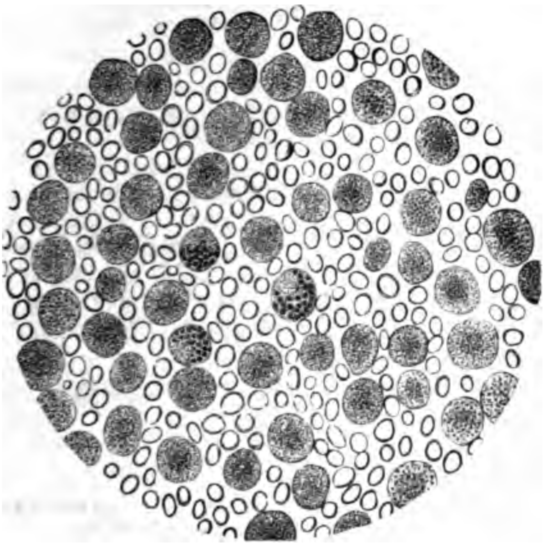


FIG. 50.—Blood from a case of pure lienal leukemia in a woman 27 years old; magnified 240 times (personal observation, Zurich clinic).

Should considerable amounts of blood be available, not only will the marked *pallor of the blood* frequently be evident to the unaided eye, but *gray and grayish-white coagula* readily form, which on microscopic examination will be found to consist principally of colorless blood-corpuscles.

More exact *physical and chemic examination of the blood* will disclose a number of other profound and significant alterations, but these are of subordinate importance to the general practitioner. Leukemic blood retains its *alkaline reaction*, although on standing in the air the reaction often becomes acid with unusual rapidity. The *specific gravity of the blood*, which normally is between 1056 and 1058, is not rarely lowered. Abnormal metabolic products occur in the blood, which appear to be swept into the circulation from the blood-forming organs. Hypoxanthin is believed to be derived principally from the spleen, glutin from the bone-marrow. In addition, xanthin, lecithin, formic acid, lactic acid, succinic acid, peptone, deuteroalbumose, leucin, and nuclein-phosphoric acid may further be mentioned. On standing in the air crystals in the form of double pyramids—*Charcot-Neumann crystals*—frequently form, which here and there are attached to colorless blood-corpuscles and appear to be derived from them (Fig. 51).

As has already been indicated, the picture presented by the blood in cases of leukemia is a variable one, and in the course of the disease the colorless blood-corpuscles progressively increase in number, while the red blood-corpuscles and, in accordance therewith, the percentage of hemoglobin, steadily diminish. Nevertheless, periods occasionally occur in which the condition of the blood improves, and particularly on the appearance of febrile infectious diseases the number of colorless blood-corpuscles has been observed to diminish considerably.

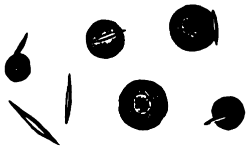


FIG. 51.—Leukemic crystals from the blood, partly free, partly contained in colorless corpuscles (after v. Zenker).

Among the *changes in the blood-forming organs, enlargement of the spleen* occurs most frequently, because in the earlier clinical sense lienal leukemia is the most frequent variety of the disease. The spleen not rarely attains such considerable proportions that it occupies not only the entire left, but also the greater part of the right half of the abdominal cavity, displacing and compressing the remaining abdominal viscera, and by pushing the diaphragm, the lungs, and the heart upward, induces dyspnea, palpitation of the

the abdomen becomes greatly increased, the patient assumes an attitude with the body inclined backward, and in several instances careless observers have mistaken leukemia for pregnancy.

In cases of *lymphatic leukemia enlargement of the external and internal lymphatic glands* takes place. Nodular packets of lymphatic glands, often as large as a fist, appear in the axillæ, the groins, and the neck, giving rise to visible deformity, and on palpation feeling at times rather like soft and at other times like hard nodules. Generally they are not tender on pressure. The overlying skin is usually tense, but otherwise unaltered. Rarely caseation and suppuration take place. Further, variations in the size of the masses of lymph-glands take place occasionally in the course of the disease. In searching for *enlargements of internal lymphatic glands,* dulness over the manubrium sterni and along the vertebral column should especially be looked for, in the situations where the lymphatic glands occur in the anterior and the posterior mediastinum; and also palpable nodular tumors in the abdominal cavity.

In rare instances only the internal lymphatic glands have become enlarged. Further, considerable enlargement of the *lymph-follicles of the intestinal mucous membrane,* of the *tonsils,* and of the *glands at the base of the tongue* occasionally takes place.

In the demonstration of *involvement of the bone-marrow,* importance was formerly attached to pain in the bones induced by pressure. Such pains are encountered especially over the sternum and a number of long bones, particularly the femora. The symptom is unreliable and may be present in the absence of disease of the bone-marrow. The occurrence of yielding points over the sternum is more valuable. The most certain diagnostic criterion, however, is afforded by examination of the blood, from the presence of nucleated red corpuscles, polynuclear eosinophile cells, mast-cells and myelocytes. In addition to the changes in the blood and in the blood-forming organs, certain *metabolic disturbances* are important in the diagnosis of leukemia. *Increased elimination of uric acid* has been recognized for some time, the amount being increased absolutely or only in proportion to the amount of urea. In addition, according to recent investigations, there is an increased elimination of the alloxuric bodies in general through the urine. Further, the alterations in question do not occur unexceptionally. Their occurrence is attributed to the fact that many colorless blood-corpuscles are destroyed and that the substances in question are formed in increased amount from their nuclein-bodies.

The *onset of leukemia* is generally so insidious that it is but seldom possible to determine it with any degree of certainty. The patients, as a rule, consult a physician only after they have become exceedingly pale or after their strength has greatly failed, so that they tire quickly even from light work, become short of breath, sweat profusely, and suffer from palpitation of the heart. Some present themselves with a progressively enlarging abdominal tumor, others on account of the deforming enlargement of the lymphatic glands. Often **evening elevation of temperature** takes place. The **appetite** is frequently impaired and **thirst** is increased. In the course of the disease two series of phenomena especially become conspicuous—in the first place, a **tendency to hemorrhage**, and, in addition, a **tendency to inflammation.** *Hemorrhage* may take place from any organ. It contributes to the progressive anemia and exhaustion, and through frequent recurrence and persistence occasionally causes death from loss of blood. At times smaller or greater hemorrhages beneath the skin are encountered; at other times there may be profuse epistaxis or bleeding from the gums; but serious hemorrhage from the air-passages, the stomach, the intestines, the urinary passages, and the generative organs has also been observed. Not rarely *cerebral hemorrhage* occurs, terminating fatally within a short time amid the symptoms of an apoplectic attack. Among the *inflammatory lesions, leukemic stomatitis* may be mentioned first, and also *leukemic retinitis.* The latter will be disclosed by the pale-yellow color of the fundus of the eye on examination with the ophthalmoscope. The retinal veins are occasionally conspicuous from the presence of broad, white marginal bands, and here and there hemorrhages and white spots are visible. The optic papilla is at times reddened and swollen.

Occasionally complications arise as a result of *pressure by enlarged lymphatic glands* or of the presence of *lymphomata.* Thus, bronchostenosis occasionally develops, or paralysis of the recurrent nerve, or palpitation of the heart in consequence of pressure upon the vagus, or difficulty in deglutition may be present. At times facial paralysis or auditory paralysis has occurred in consequence of compression and paralysis of the respective nerves by lymphomatous new-growths. Naturally, disturbances of hearing and the symptoms of Ménière's disease not rarely attend hemorrhage into the labyrinth. Lymphomata in the orbit have given rise to exophthalmos. In a patient under my care spinal compression-paralysis resulted from the presence of lymphoma in the peridural fatty tissue of the vertebral canal. Nodules and nodular lymphomata also occur occasionally upon the skin, at times causing ulceration. Frequently the *liver* becomes enlarged, and this change also is dependent upon the presence of lymphomata and interlobular connective-tissue hyperplasia. Obstinate *priapism* has been observed repeatedly in consequence of the formation of thrombi from colorless blood-corpuscles in the blood-spaces of the corpus cavernosum of the penis.

Leukemia pursues a *chronic course,* as a rule, although cases of *acute leukemia* also occur, terminating fatally within a few days or weeks. The duration of chronic leukemia is usually from one to two years. Death results most frequently from progressive exhaustion, hemorrhage, or intercurrent disease.

Diagnosis.—The recognition of leukemia is generally easy. It is distinguished from *leukocytosis* by the fact that in the latter the increase in the number of colorless blood-corpuscles is but transient and generally also slighter, and that only the leukocytes present in normal blood are encountered. It should be observed that the blood during the *death-struggle* not rarely resembles that of leukemia, although, under such conditions, the large number of colorless corpuscles present in the blood depends upon inequality in distribution in the presence of circulatory disturbances. Leukemia, in common with **pseudoleukemia,** is attended with enlargement of the lymphatic glands and the spleen, although the colorless blood-corpuscles are not increased in pseudoleukemia.

Anatomic Alterations.—In the **blood,** especially the blood of the cavities of the heart, grayish or yellowish coagula, constituted principally of white blood-corpuscles, are often conspicuous, and occasionally on opening the cavities of the heart an impression is created as if an abscess had been incised. The **lymphatic glands** and the **spleen** not rarely attain an enormous size. In the spleen the follicles are not rarely converted into large gray nodules. The lymphatic glands on section generally present a grayish-red color, and their tissues are at times dense, and at other times soft and juicy, accordingly as hyperplasia of the trabecular or of the lymphadenoid cells preponderates. Often, though not unexceptionally, the fatty marrow of the **long bones** is wanting, being replaced by the grayish and pus-like tissue—*pyoid bone-marrow*—or by red tissue, suggestive of raspberry-jelly—*lymphoid* or *red bone-marrow*. The first consists of polynuclear cells with an abundance of protoplasm, and the latter of mononuclear cells with a small zone of protoplasm. On exposure to the air Charcot-Neumann crystals are frequently separated in large amount from the spleen and the bone-marrow. On puncture of the spleen it has further been found that the crystals named are present in this organ even during life. Often almost all of the viscera are the seat of *lymphomata,* which occur either as nodules or as diffuse infiltrates. The latter occur as thick cushions especially in the pleural, peritoneal, and pericardial cavities. The lymphomatous nodules are visible here and there to the unaided eye, or they can be detected only with the aid of the microscope. Also in the blood-vessels of the **brain,** in addition to thrombi of colorless blood-corpuscles, collections of colorless blood-corpuscles have been observed in the adventitial sheaths and in the vicinity of the blood-vessels. Necrotic areas of softening and extensive columnar degeneration have been observed in the **spinal cord.**

Prognosis.—Leukemia is an incurable disease, and the prognosis is therefore unfavorable. In the exceedingly rare cases in which recovery has been reported it is probable that errors in diagnosis were made.

Nothing is known as to the *nature of leukemia.* Efforts have been made to attribute the disease to *bacteria* or to *auto-intoxication*, but all evidence is wanting for these hypotheses. In all probability the disease results from the overwhelming of the blood with colorless corpuscles in consequence of morbid activity of the blood-forming organs. In addition, colorless blood-corpuscles are destroyed in larger number, with an increase in the amount of uric acid and the alloxuric bodies, and, at the same time, there is deficient regeneration of red blood-corpuscles.

Treatment.—**Causal therapy** may be resorted to especially if malaria or syphilis has preceded the development of leukemia, but *quinin*, *eucalyptus*, and similar remedies in the first, as well as mercurials and iodids in the second instance, are generally without effect. In **symptomatic treatment** the greatest importance should be attached to a light, nutritious *diet* and *life in the open air*. Among medicaments, especially those have been employed to which some influence upon blood-formation has been attributed, especially **iron, arsenic,** and **phosphorus.** I have observed transitory good results from the use of arsenic given in large doses and for a long time:

℞ Solution of potassium arsenite,
Bitter-almond water, each, 5.0 (75 minims).—M.
Dose: 10, 15, or 20 drops in a half-wine-glassful of water thrice daily after meals.

Some patients under my care bore subcutaneous injections of arsenic badly, while they did well upon internal administration of the same remedy.

Inhalations of oxygen have been recommended by some physicians, although they have not always been successful.

The *enlarged spleen* has often been made the point of attack in treatment, and ice-bags, douches, electropuncture, injections of so-called splenic remedies (arsenic, ergotin), faradization, and massage have been employed. Resort has even been had to splenectomy, although this must be considered a mistake, for almost without exception death from hemorrhage has resulted either during the operation or shortly thereafter. In addition, no effect whatever upon the leukemia is to be expected if the view is correct that the spleen is never involved primarily, but always secondarily in cases of leukemia. Extirpation of enlarged lymphatic glands is scarcely ever to be thought of, in view of the extent of the changes. Recently **specific treatment** has been attempted. This includes *organotherapy*, and, in accordance with the variety of leukemia, spleen, lymphatic glands, or bone-marrow has been prescribed in the form of tablets or of fresh tissue. I have personally observed no permanent result from organotherapy. It should be mentioned further that, upon the basis of the experience that the number of colorless blood-corpuscles is frequently diminished in conjunction with intercurrent infectious diseases, *injections of antistreptococcic serum* have been recommended. In a patient under my observation suppuration of a number of lymphatic glands developed as a result of such treatment, while the state of the blood remained unaltered.

PSEUDOLEUKEMIA.

Etiology.—Pseudoleukemia derives its name from the fact that it resembles leukemia with regard to the *enlargement of the blood-forming organs* and the *progressive anemia*, although, on the contrary, abnormal increase in the number of colorless corpuscles in the blood does not take place. Increased elimination of alloxuric bodies in the urine is therefore also wanting. With regard to the causes of pseudoleukemia but little of a definite nature is known. Experience has shown that *men* are attacked by the disease more commonly than women, and the disorder generally occurs between the *twentieth and the sixtieth year of life*. It is encountered more frequently among the well-to-do than among the *poorer classes* in the community. Occasionally the affection is attributed to **antecedent infectious disease**, especially malaria, syphilis, and scrofulosis. **Chronic inflammatory processes** also, as, for instance, of the lacrimal duct or of the ear, are believed to be the point of origin for the disorder; and, likewise, chronic diarrhea. **Alcoholism** and **rachitis** also have been considered as causes. Pseudoleukemia is a rather *uncommon disease.*

Symptoms.—**Alterations in the blood-forming organs** are generally placed among the earliest symptoms, and pseudoleukemia also is subdivided into *lymphatic*, *lienal*, and *myelogenic* varieties, accordingly as swellings and changes take place in one or another of the respective organs exclusively or earliest. Little, it is true, is known with regard to myelogenous pseudoleukemia. Just as in the case of leukemia, *mixed varieties* of pseudoleukemia also occur frequently. In cases of *lymphatic pseudoleukemia* the glands of the post-cervical region, the submaxillary region, the axillary region, and the inguinal region are generally involved earliest, but the mediastinal and the peritoneal lymphatic glands also are involved. Not rarely packets of enlarged lymphatic glands larger than a fist have developed in various portions of the body, giving rise to deformity and functional disturbances. At times the glands are rather soft, and at other times rather dense in consistency. It is especially in cases of the first kind that the enlargement is particularly marked and takes place rapidly. The affected lymphatic glands are but little, if at all, tender on pressure. The overlying skin is generally unaltered and movable. Inflammation and suppuration, as well as caseation, of the glands occur but rarely, although at times enlargement and diminution in size take place in the course of the disease. The *spleen* frequently acquires the same size as in leukemia, and gives rise to similar symptoms as in that disease. Involvement of the *bone-marrow* will be suspected from yielding points in the bones rather than from pain on pressure.

Generally the symptoms mentioned have developed so insidiously that it is impossible to determine with certainty the commencement of the disease. The beginning of the *anemia*, likewise, can generally not be determined. The patients grow more and more pale, become short of breath, and tire and perspire readily, even after slight physical exertion. If a drop of *blood* from the tip of the finger or the lobe of the ear be examined, its pale-red color will frequently attract attention. The number of red blood-corpuscles and the percentage of hemoglobin are occasionally reduced to one-fifth of the normal. The colorless blood-corpuscles are not increased in number and exhibit no unusual forms. Not rarely protoplasmic granules (elementary granules) are unusually numerous. The red blood-corpuscles not infrequently exhibit polymorphism (poikilocytosis). The *circulatory apparatus* presents anemic alterations, thus, not rarely, systolic heart-murmurs, dilatation of the right ventricle, palpitation of the heart, venous murmurs in the neck, etc. Frequently the patient complains of impaired appetite and increased thirst. The urine exhibits no noteworthy change. Spontaneous hemorrhage occurs less commonly than in cases of leukemia.

The *course of pseudoleukemia* is generally chronic, and occasionally extends over several years. Often the disease is unattended with fever, while in other instances irregular *febrile movement* occurs, particularly in the evening; and at times afebrile and febrile periods have been observed to alternate with each other at approximately regular intervals.

Among *complications pressure-phenomena* induced by enlarged lymphatic glands are especially to be mentioned, as, for instance, bronchostenosis, paralysis of the recurrent laryngeal nerve, neuralgia, particularly jaundice and enlargement of the liver, so that in the presence of the last-named symptoms the disease may resemble hypertrophic cirrhosis of the liver to such a degree as to be mistaken for it. At times *amyloid degeneration* of various organs also develops in the course of the disease. Occasionally cutaneous alterations occur, particularly *prurigo*, which is probably dependent upon the presence of lymphomata in the skin.

Death results most frequently from progressive *exhaustion;* less commonly from *uncontrollable hemorrhage* or accidental inter-
nt disease. In some cases pseudoleukemia has been observed
ually transformed into *leukemia*.

Diagnosis.—The differentiation between pseudoleukemia and **leukemia** is easy, for the former is unattended with increase in the number of colorless corpuscles in the blood. There is, also, generally no difficulty in distinguishing lienal pseudoleukemia from other **chronic enlargement of the spleen** (malaria, syphilis, amyloid). The anemia attending the latter is less marked, and is without the serious significance it has in the former. The differentiation between lienal pseudoleukemia and **hypertrophic cirrhosis of the liver** is occasionally impossible, and even upon post-mortem examination the two diseases so closely resemble each other that the lymphomata distinctive of pseudoleukemia are discovered only upon microscopic examination of the viscera. Lymphatic pseudoleukemia may, in the first place, be readily confounded with **tuberculosis of the lymphatic glands**, and in the differentiation the greatest importance is to be attached to the detection of tuberculosis in other organs, and especially in the lungs, in order to decide in favor of tuberculosis of the lymphatic glands. Likewise the diagnosis would be rendered certain by the detection of tubercle-bacilli in the secretion from suppurating lymphatic glands, or in the tissue excised from lymphatic glands. In addition, confusion between lymphatic pseudoleukemia and **lymphosarcomatosis** may readily arise, and it must be admitted that at the present time no sharp clinical or anatomic distinction between the two diseases can be made. Soft enlargements of the lymphatic glands have in many instances been considered sarcomatosis of the lymphatic glands. Perhaps a clearer differentiation will become possible when an explanation is forthcoming as to the as yet wholly unknown *nature of the disease*. Some clinicians have also assumed *infectious*, and others, again, *autotoxic influences*, in the development of pseudoleukemia, but these hypotheses are without substantial basis. Nothing is gained by the assumption that pseudoleukemia is a *premonitory stage of leukemia*, as nothing also is known with regard to the conditions that lead up to leukemia, apart from the fact that serious objections may be raised against the opinion in question.

Anatomic Alterations.—The anatomic alterations in the lymphatic glands, the spleen, and the bone-marrow correspond with those of leukemia, and the correspondence extends to the presence, in cases of pseudoleukemia, of macroscopically or only microscopically visible lymphomata in various organs. The liver is enlarged and studded with lymphomatous nodules with especial constancy. Enlargement of the periportal lymphatic glands has not rarely given rise to jaundice through pressure upon the large biliary ducts.

Prognosis.—Pseudoleukemia is generally an incurable disease, although marked transient improvement can now and again be obtained.

Treatment.—The treatment of pseudoleukemia is identical with that of leukemia (pp. 246 and 247), and I have obtained good results especially from the use of arsenic and iron:

℞ Tincture of ferric chlorid,	20.0 (5 fluidrams);
Solution of potassium arsenite,	10.0 (2½ ").—M.

Dose: 20 drops thrice daily after meals.

PROGRESSIVE PERNICIOUS ANEMIA.

Etiology.—Progressive pernicious anemia depends upon **insufficient and defective formation of red blood-corpuscles,** which may become so pronounced that death results from impoverishment of the blood. The disease is *not common*, but occurs more frequently in some countries, as, for instance, Switzerland, than in others. Generally it occurs in *adults*. It is more frequent among the *poorer classes* of society than among the well-to-do. Occasionally the disorder arises without demonstrable cause, and under such circumstances the designation *idiopathic, essential, primary,* or *cryptogenetic progressive pernicious anemia* has been employed. In other instances antecedent causative conditions have been present, and then the disorder is designated *deuteropathic, secondary progressive pernicious anemia*, but the etiologic factors are of so slight a character that most individuals withstand them without serious consequences, and only a few develop the profound disease of the blood in consequence. A certain **predisposition,** whose mode of origin and nature are as yet unknown, is accordingly necessary in order that pernicious anemia may develop, and the causes about to be mentioned have perhaps only the significance of provocative influences. Among these, *emotional disturbances, excessive physical activity,* and *deficient nourishment* may be mentioned. Not rarely pernicious anemia is observed in Switzerland among women employed in factories, who are compelled to work uninterruptedly in close rooms from early in the morning till late at night, and sustain themselves solely with coffee, bread, and potatoes. At times pernicious anemia develops as the result of **chronic wasting discharges,** as, for instance, after repeated epistaxis, chronic diarrhea, or leg-ulcers. Occasionally the disorder has been observed to develop after **infectious diseases,** such as typhoid fever and syphilis. **Pregnancy** and **parturition** have an unmistakable influence upon the disease, and this fact explains the preponderance of the affection among females. Even in localities in which the disorder especially prevails, periods in which many cases occur lternate with other periods in which the disease is rarely observed, [illegible]hout any explanation for the difference being forthcoming.

[illegible]ptoms and Prognosis.—The most conspicuous symp- nicious anemia is the extremely marked **pallor,** the rarely exhibiting a death-like whiteness, and even the conjunctiva, the mucous membrane of the lips and the mouth being almost wholly without a red tint. Either the patient has himself been struck by the pallor, or his attention is called to it by his friends. If the **blood** be examined, it will be found to exhibit no peculiarity of appearance, so that at this time the disease cannot be recognized with certainty from the alterations in the blood, for the same changes occur also in other varieties of profound anemia. The blood often flows freely from a puncture at the tip of the finger, although it presents a pale color, and frequently exhibits little tendency to coagulation.

On microscopic examination the *red blood-corpuscles* will be found widely separated, and generally not forming rouleaux, and from this it may be correctly concluded that they are diminished in number. Enumerations with the Thoma-Zeiss hemocytometer have shown that the number may fall to one-tenth of the normal. Subsequently the red blood-corpuscles exhibit great polymorphism —*poikilocytosis*. Cells of normal shape alternate with pear-shaped, figure-of-eight-shaped, dumb-bell-shaped cells, and others shaped like the root of a tooth (Fig. 52). At times *ameboid movements* may be visible in some red blood-corpuscles. *Variability in the size* of the red blood-corpuscles is noteworthy. Together with red corpuscles of excessive size—giant blood-corpuscles, megalocytes, gigantocytes—exceedingly small red blood-corpuscles—microcytes—are encountered, which at times are as diminutive as small

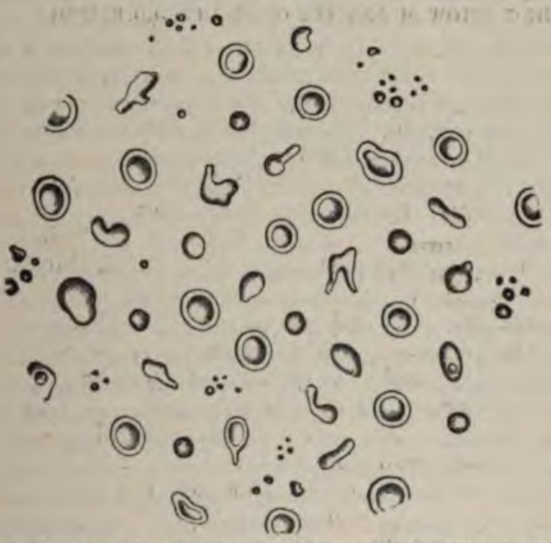


FIG. 52.—Blood from a case of progressive pernicious anemia; poikilocytosis and isolated globular microcytes; magnified 600 times (personal observation).

drops of hemoglobin. Two varieties of microcytes should be distinguished, the concave and the globular. While some consider the microcytes as imperfectly developed red blood-corpuscles, others attribute them to constriction of ordinary red blood-corpuscles, and therefore designate them *schistocytes*, and endeavor to explain their occurrence as an effort to increase the oxygen-absorbing surface of hemoglobin. *Nucleated red blood-corpuscles* are unexceptionally encountered. These are often as large as ordinary red blood-corpuscles, and are then designated *normoblasts.* The occurrence, however, of large nucleated red blood-corpuscles—*megaloblasts*—is especially important, indicating abnormal activity of the bone-marrow suggestive of embryonal activity.

Stained, dry preparations of the blood disclose that also the structure of the red blood-corpuscles has suffered in cases of pernicious anemia, inasmuch as on treatment with a solution of eosin and hematoxylin they assume in part a bluish tint rather than the reddish tint of eosin. This peculiarity has been designated *polychromophilia.* Further, it will be found occasionally in fresh blood-preparations that the hemoglobin has in places separated from the stroma of the blood-discs. In isolated observations also peculiar brownish or yellowish-red blood-corpuscles have been described.

In conformity with the diminution in the number of red blood-corpuscles there occurs also a *reduction in the percentage of hemoglobin*, which may reach 15 per cent. of the normal. The statement that the percentage of hemoglobin is diminished in lesser degree than the number of red blood-corpuscles, and that it is distinctive of pernicious anemia that some red blood-corpuscles contain an excessive amount of hemoglobin, I am unable to confirm as a rule from personal observation. The *colorless blood-corpuscles* are rather diminished (hypoleukocytosis), and exhibit no peculiarity in shape. *Blood-plates* and *elementary granules* are frequently increased in considerable degree.

Some observers have described *lower forms of organisms* (flagellates, leptothrix, protozoa) in the blood, although these statements are not above criticism. *Physical and chemical examination of the blood* discloses that the specific gravity is considerably diminished (the normal is 1058), and that, accordingly, the blood contains considerable water and little solid matters, particularly albumin and nitrogen. The alkaline reaction of the blood is almost unchanged. If the blood be preserved in a glass cylinder and sedimentation is permitted to occur, the lowermost layer, constituted of red blood-corpuscles, is a very small one. The *total amount of blood* has been observed to be reduced one-half.

As may be expected, profound anemia is attended with various functional disturbances. Above all, fatigue readily takes place as a result of any physical exertion, and the patient generally complains at the same time of palpitation of the heart, dyspnea, and sweating. Occasionally these represent the first symptoms of the disease. While the muscles are wasted and small, the layer of *subcutaneous fat* is generally not merely preserved, but is occasionally developed excessively. At times some of the **bones**, with especial frequency the sternum, are exceedingly tender on pressure. Often **cutaneous edema** develops, probably in consequence of defective nutrition of the smaller blood-vessels, and resulting in abnormal permeability of the vessels to the blood-plasma. At times, and especially at the beginning, there is edema of the ankles, and at other times the face especially is puffy, edematous, and distorted. I have observed also edema of the conjunctiva—chemosis—in a number of instances.

The **bodily temperature** is in some cases unaltered, but often febrile movement appears, following no definite type, and the temperature occasionally attains so high a level that the clinical picture of typhoid fever is suggested. Under such circumstances the designation *anemic fever* is frequently employed, by which it is intended to indicate that the fever may be the result of disturbances in the activity of the heat-regulating centers in consequence of the anemia. Other symptoms of **disturbed cerebral activity** also occur not rarely in cases of pernicious anemia. The patients often suffer from obstinate *sleeplessness—agrypnia.* Others, however, lie for days and weeks in a somnolent and dreamy state, indifferent to their surroundings, and now and again muttering unintelligible words and sentences. It may also happen that the patient sleeps quietly by day, but becomes restless with the approach of night, throwing himself about, speaking aloud, and even crying, and becomes a nuisance to his friends in consequence of his noisy and excitable state. *Disinclination to mental activity* and *impairment of memory* become notable in the psychic sphere. Occasionally wild *delirium* and *maniacal states* occur. The patient frequently complains of *vertigo, headache,* a *tendency to syncopal attacks, roaring in the ears, obscuration of the field of vision,* manifestations that occur especially on assuming the erect posture. **Spinal symptoms** also occur in some patients, especially paresthesiæ, hyperesthesia, and anesthesia, paresis and abolition of the knee-jerks, unsteadiness in standing, and even immobility of the pupils.

Anemic symptoms referable to the circulatory apparatus are encountered with especial constancy, including systolic, rarely also diastolic, heart-murmurs, dilatation of the right ventricle, palpitation of the heart, venous murmurs in the neck, negative venous pulse, arterial murmurs over the bulb of the femoral vein. The lungs and the **air-passages** generally remain unaffected. Such shortness of breath as may be present is generally dependent upon the anemia, and but rarely upon disease of the respiratory organs. The **appetite** is, as a rule, wanting. Rarely *excessive appetite—bulimia*—occurs. *Thirst,* however, is generally ncreased. The patients not rarely give off a penetrating odor from the mouth. On *examination of the stomach* absorption will generally be found to be retarded, and motor activity diminished, together with an absence of free hydrochloric acid from the gastric juice. The **bowels** are generally constipated, although occasionally obstinate diarrhea occurs. The **liver** but rarely presents alterations, but now and then slight *icterus* appears. **Enlargement of the spleen** is frequently but not constantly present. The **urine** is not rarely of a dark color, and contains an abundance of nitrogen, urea, and indican. Frequently it contains small amounts of albumin. Marked nephritis occurs but seldom.

Among the most frequent *complications* of progressive pernicious anemia are *hemorrhages,* which may take place in all possible organs. Preferably they have a punctate distribution, but they readily become confluent, with the development of the effects of a massive hemorrhage. Obviously the hemorrhage is in part the result of abnormal permeability and in part of increased fragility of the vessels. Hemorrhages into the external integument are without significance, while *epistaxis* may be attended with serious dangers by reason of its copiousness, resistance to control, and frequent recurrence. Occasionally bleeding from the gums takes place. Hematemesis, hematuria, enterorrhagia, metrorrhagia, are among the less common occurrences. Occasionally symptoms of *cerebral hemorrhage* appear, in consequence not of massive extravasation of blood, but of capillary bleeding, which, by reason of the large number and the close proximity of the extravasations, acquires the characteristics of a massive hemorrhage. *Retinal hemorrhages,* which are not rarely found in considerable number on ophthalmoscopic examination, occur with especial frequency, and are therefore of diagnostic significance. At times they are rather circular in shape, at other times of longitudinal form, and in the latter event they often radiate toward the center of the optic papilla. Occasionally their center is of a yellowish color, but yellowish spots appear also in the retina apart from hemorrhages. Neuroretinitis also has been observed. The patients under such circumstances often do not complain of the slightest disturbance in vision.

Progressive pernicious anemia generally pursues a *chronic course,* and occasionally extends over more than a year. Often numerous exacerbations and remissions occur, but in most cases death eventually results in consequence of excessive blood-impoverishment. Some physicians consider permanent recovery impossible, and they contend that recovery even for as long as a year
always only apparent, and will be followed by a fatal recur-
ears to be going too far. Naturally, the *prog-*
stances; nevertheless we have

Anatomic Alterations.—As among the symptoms, so also among the anatomic alterations, no single one can be named that is distinctive of the disease. The viscera exhibit only the appearances resulting from profound anemia, namely, *pallor, anemic fatty degeneration, hemorrhage,* and the *presence of iron in large amount* (*siderosis*). The **fatty layer of the skin** frequently is exceedingly well developed, and often is characterized by a deep sulphur-yellow color. The **muscular tissue,** however, is generally pale, soft, and wasted, and on microscopic examination exhibits granular turbidity, fatty degeneration, occasionally also vitreous degeneration, and abundant pigment-deposition.

The **heart** is almost wholly empty, and such blood-clots as may be present are pale in color and soft in consistence. Often the subepicardial fatty tissue is present in large amount. The myocardium is pale, often light brown, and even to the unaided eye exhibits signs of most marked fatty degeneration. Particularly on the papillary muscles, but also upon the heart-wall proper, numerous butter-yellow points and lines are visible, which, on microscopic examination, are found to correspond with myocardial fibers involved in fatty degeneration. Often the points and lines have become confluent, and impart to the myocardium a speckled, marbled, or mottled appearance. In addition, hemorrhages not rarely occur in the myocardium, most frequently of a punctate character. The *endocardium* is generally delicate, and occasionally slightly icteric. The **aorta** also frequently is noteworthy for the thinness of its walls and its distensibility. The **serous cavities** almost always contain transudates, which occasionally present a slightly bloody or icteric tint. The **lungs** and the **air-passages** are characterized by pallor, and often exhibit numerous punctate, rarely larger, extravasations of blood. Pulmonary edema is often present, and the laryngeal mucous membrane also is swollen and edematous in some cases. The **spleen** frequently is increased in size, but generally is of dense consistency. The presence in it of an excess of iron is readily demonstrable by microchemic methods.

The **liver,** however, is generally unchanged in size, but presents an ochre-yellow or rusty-yellow color, due to the presence of an excessive amount of iron-granules. On microscopic examination the liver-cells will be found involved in a more or less marked degree of fatty degeneration, and, in addition, the presence of the iron-granules in unusually large amount can be demonstrated by means of certain *iron-reactions.* If, for instance, thin sections of liver are introduced into a solution of ammonium sulphid, they will generally acquire a smoky-gray color, appreciable macroscopically, while on microscopic examination the liver-cells will be found filled with minute black iron-granules. Another iron-reaction consists in the introduction of thin sections of liver, first into a solution of yellow potassium ferrocyanid, and then in dilute hydrochloric acid. The sections become blue, and on microscopic examination all iron-granules are found stained blue. The large amount of iron present in the liver has been demonstrated also by chemic means. The **bile** is generally dark in color.

The **gastric** and the **intestinal mucous membrane** exhibits on microscopic examination marked *glandular atrophy*, and the former especially resembles, in many instances, a granulating surface. The muscular layer of the stomach and the intestine exhibits here and there fatty degeneration and the presence in individual muscle-fibers of brownish pigment-granules. Such glandular cells as are still present are generally in a state of granular cloudiness or fatty degeneration. The **kidneys** are pale, and often exhibit small hemorrhages. Microscopic examination will disclose granular clouding and fatty degeneration of the epithelial cells of the convoluted uriniferous tubules. If the iron-reaction be applied, an abundance of iron-granules will be found also in the kidneys, particularly in the epithelial cells of the convoluted uriniferous tubules. The **mucous membrane of the urinary passages** is frequently edematous and swollen, and the seat of punctate hemorrhages. The *marrow* of the long bones of the extremities has often lost its yellow, fatty appearance, and has acquired an appearance suggestive of raspberry-jelly—so-called *lymphoid bone-marrow*. On microscopic examination it is characterized by the large number of nucleated red blood-corpuscles present, particularly megaloblasts, but also microcytes are present in large number.

Frequently changes in the **nervous system** take place. Punctate hemorrhages occur on the inner aspect of the *cerebral dura mater*, and new membranes resulting therefrom constitute an almost constant feature. Through the transformation of the hemoglobin into hematoidin the inner aspect of the dura acquires at times a yellowish, icteric appearance. In the **brain** the white matter especially is occasionally the seat of numerous punctate hemorrhages, which in places lie close together, and appear to constitute a single large lesion. Punctate hemorrhages are of frequent occurrence also in the white matter of the **spinal cord**. These not rarely cause swelling and degeneration of small groups of nerve-fibers in the immediate vicinity. Nevertheless, independently of antecedent hemorrhage, small areas of softening occur in the white matter of the cord, in which the axis-cylinders are swollen and enlarged, and the medullary sheaths have undergone destruction, alterations that appear to be the result of a deficient supply of blood. It is, however, noteworthy that columnar degeneration in the spinal cord may also take place. The posterior columns of the cord especially exhibit a tendency to such alterations, which correspond with tabic symptoms during life. Hemorrhage and degenerative alterations occur occasionally in the **peripheral erves.**

Diagnosis.—The recognition of progressive pernicious anemia is exceedingly difficult because as yet no distinctive symptom is known, so that the diagnosis can never be made with absolute certainty. Even the experienced clinician may, therefore, not rarely fall into error. The disorder may be readily confounded with other states of profound anemia, and above all carcinoma and intestinal worms must be taken into consideration. In the *differential diagnosis from carcinoma* the detection of a tumor especially is important, in addition to the *age* of the patient, but carcinomata are frequently concealed. In cases of carcinoma of the stomach, for instance, the derangements in the functions of that viscus (delayed absorption, diminished motor activity, absence of free hydrochloric acid in the gastric juice) agree entirely with those of progressive pernicious anemia. The blood-findings in both diseases are also the same. As a rule, retinal hemorrhages occur less commonly, and in smaller number, in cases of carcinoma of the stomach. Elevation of temperature also is usually more transient, slighter, and less common. *Intestinal worms*, particularly the Bothriocephalus latus, the Ankylostomum duodenale, the Ascaris lumbricoides, even the Bacterium termo, are capable of causing similar anemic states as are encountered in cases of pernicious anemia, although the parasites named or their ova will be found in the intestinal evacuations, and, besides, the patients regain their former state of health after expulsion of the parasites. Pernicious anemia is distinguished from profound chlorosis by the fact that the former is generally unattended with fever and retinal hemorrhages, and is quickly cured by preparations of iron. Primary atrophy of the lab-glands and the intestinal glands is also mentioned as a source of error, although we are as yet not convinced that this disorder may arise in an independent manner.

Nothing of a definite nature is known with regard to the *nature of progressive pernicious anemia.* In accordance with prevailing views some clinicians have assumed a *bacterial origin* for the disease, while others are inclined to admit its *autotoxic development* from the stomach and the bowel. The reports with regard to the presence of bacteria in association with the disease are most untrustworthy. In support of the autotoxic nature of the disorder it has been pointed out that recovery speedily takes place in some cases after irrigation of the stomach. The *seat of the disease* has been referred to the bone-marrow, and it has been believed that the abundance of megaloblasts in the marrow and the blood favors the view that the process of blood-formation is interfered with and, to a certain degree, has reverted to the *embryonal type.* In our opinion, not only blood-formation but also *blood-destruction* has undergone a morbid alteration, in consequence of which the probably less resistant red blood-corpuscles undergo degeneration in exaggerated degree. The dark urine, rich in urobilin, frequently observed, the increased nitrogenous elimination through the urine that can often be demonstrated, the unusually dark-colored bile, and the large amount of iron in many organs, seem to be in favor of this view.

'atients with progressive pernicious anemia
itive, but at the same time easily digestible

diet, especially milk, and should live largely in the open air, in reclining chairs or in hammocks. Among drugs, **arsenic** may be considered a specific, and from its long-continued use recovery has been observed in a number of instances.

℞ Solution of potassium arsenite, 10.0 (2½ fluidrams);
Tincture of ferric chlorid, 20.0 (5 ").—M.
Dose: 20 drops thrice daily after meals.

Other hematinics also have been employed, especially *preparations of iron* and of *phosphorus*. *Blood-transfusion* and *blood-infusion* and *preparations of hemoglobin* also have been resorted to. Recently recourse has been had to *organotherapy*, and bone-marrow has been prescribed.

If syphilis is believed to be the cause of the disease, good results may be obtained through **specific treatment** (iodin, mercury). **Irrigation of the stomach and the intestines** and the internal administration of **salol** also are thought occasionally to have induced surprising results through disinfection of the gastro-intestinal tract.

CHLOROSIS.

Etiology.—Chlorosis is attended with a **deficiency in the hemoglobin of the blood.** The disease is extremely *common*, and occurs especially, and almost exclusively, in women. It develops but seldom in childhood. Most frequently it commences at the period of puberty, and only exceptionally beyond the thirtieth year of life. A distinction can be made between *hereditary* and *acquired chlorosis*. Most commonly the condition is one of *hereditary chlorosis*, but also this occurs often in the sequence of those injurious influences that are themselves sufficient to induce chlorosis in individuals without hereditary predisposition. In the first place, excessive **sedentary occupation indoors** should be mentioned as particularly injurious. The disorder is therefore encountered with especial frequency in tailors, workers in factories, teachers, and students. Chlorosis will occur the more readily if, in addition to the excessive confinement indoors, **excessive physical activity** and **deficient** and **unsuitable food** are indulged in, and which are themselves competent to induce a chlorotic state of the blood. There is good ground for the belief held by some chlorotic patients that their disease is dependent upon **emotional disturbances**, among which particularly anxiety, grief, homesickness, and disappointment in love may be mentioned. Chlorosis is often attributed to **menstrual disturbances**, assuredly in many instances incorrectly, inasmuch as the menstrual derangement is not the cause, but the consequence of an already existing chlorosis. Most frequently menstruation is irregular or it remains wholly absent for a time, or it becomes scanty and painful; occasionally, however, menstrual hemorrhage is excessive. Often the appearance of chlorotic symptoms has been preceded by *gastro-intestinal dis-*

turbances, particularly loss of appetite and constipation. **Long-continued illness** and **wasting discharges** also occasionally constitute the origin of chlorosis. Profound chlorosis occurs most frequently in large cities with extensive factories, although it would be incorrect to assume that the disease is rare in agricultural communities.

Symptoms and Prognosis.—Among the symptoms of chlorosis the **pallid appearance** most frequently attracts attention; particularly the cheeks, the lips, the lobule of the ear, and the conjunctiva are of strikingly pallid tint. Often the patients do not present a pure white, but rather a yellowish-white or a greenish-white appearance; hence, the name chlorosis, from χλωρός, greenish-white. If the **blood** be examined in the usual manner, it generally escapes readily and freely from the wound in the skin, but even to the unaided eye it is conspicuous by reason of its *light-red color.* Estimations of the blood coloring-matter show that the *percentage of hemoglobin* is diminished, occasionally to as low as one-fifth of the normal. It is maintained by some physicians that the *red blood-corpuscles* are at first not diminished in number. The number of erythrocytes is thought to decline only at a later period, when chlorosis is complicated by secondary anemia. Whether these statements are correct is not certain; at any rate, I have encountered oligocythemia together with oligochromemia even in recent cases of chlorosis; but it is true that the deficiency in hemoglobin is usually more marked than the reduction in the number of red blood-corpuscles, so that *impoverishment of the individual red blood-corpuscle in hemoglobin* is a distinctive feature of chlorosis. Additional important alterations in the blood do not occur, except that chemic examination of the blood discloses a *deficiency of iron.* All of the remaining symptoms depend upon the deficiency in hemoglobin and the resulting interference with gaseous interchange in the tissues, as manifested in diminished functional activity and a tendency to deranged activity in individual organs.

The patients almost invariably complain of undue readiness of **fatigue**, and are often seized with shortness of breath on slight physical exertion, with sweating and palpitation of the heart. The entire facial expression is one of languor, and the patients avoid all movement as much as possible. Anemic disturbances occur quite constantly in the **circulatory system**, particularly objective or even only subjective palpitation of the heart, systolic anemic heart-murmurs, dilatation of the right ventricle, murmurs over the bulb of the internal jugular vein and the femoral vein, and arterial murmurs. The patients often complain of nervous symptoms, particularly **neuralgia.** Cephalalgia, hemicrania, intercostal neuralgia, and gastralgia are quite frequently sources of complaint. Occasionally complaint is made also of **roaring in the ears.** Sleep is often disturbed, or the patients are tired and sleepy by day and are able to sleep only with difficulty on the approach of night. Frequently hysteria or neurasthenia occurs as a complication of chlorosis.

Disturbances in the digestive organs are but seldom absent. The *tongue* is often coated and the patients often complain of a disagreeable *odor from the mouth.* Many patients suffer from *loss of appetite* (*anorexia*), or they have a desire for acid articles of food or even for indigestible substances (chalk, ink, and slate-pencils)—so-called *pica.* Frequently *eructations* occur, with a disagreeable and even a painful *sense of pressure in the epigastrium.* The occurrence of nervous *gastralgia* has already been referred to. Some physicians state that excessive generation of hydrochloric acid in the gastric juice—*hyperchlorhydria*—is a constant feature of chlorosis, although we are unable to confirm this statement from personal observation, but, on the contrary, the patients under our observation have more frequently exhibited *hypochlorhydria* or *achlorhydria.* Likewise the occurrence of downward displacement of the stomach (*gastroptosis*), together with a similar displacement of the right kidney (nephroptosis), must be looked upon rather as the exception than as the rule. The *bowels* are mostly constipated in consequence of anemic atony of the muscular coat of the intestine. The **urine** generally is light yellow in color, and occasionally contains small amounts of albumin—anemic albuminuria. It is further deficient in iron. **Disturbances in the generative organs** are common, with particular frequency irregular or scanty menstruation, or amenorrhea, and muco-purulent discharges.

Chlorosis is but rarely attended with serious *complications.* Slight *cutaneous edema* of anemic origin develops with relative frequency about the ankles or the eyelids, which usually soon disappears on rest in bed. Occasionally *marantic thrombi* form in the *veins*, most frequently in those of the legs, less commonly in those of the arms, at times also in the cerebral sinuses. Slight and insignificant *elevation of temperature* occurs frequently. A marked predisposition to *round ulcer of the stomach* is present.

The *course of chlorosis* is chronic. The disease is characterized by a marked tendency to *recurrence*, which in many women is repeated throughout life, and with especial regularity with the advent of spring. Some physicians believe chlorosis to be susceptible only of improvement, and not of recovery, a view that we do not ourselves consider correct. The *prognosis* of chlorosis may therefore be looked upon as favorable, as good results can often be secured within a short time.

Anatomic Alterations.—Little is known with regard to the anatomic alterations that attend chlorosis, as the patients rarely succumb to the disease. Unusual diminutiveness of the uterus, the heart, and the aorta, in consequence of deficient development—

hypoplasia—has been observed in a number of instances of hereditary chlorosis. The walls of the narrow aorta were extremely distensible, and the vessel exhibited here and there fatty degeneration of the intima, with irregularity in the origin of the intercostal arteries.

Nothing is known with regard to the *nature of the disease*. Naturally it is to be attributed to deficient blood-formation, particularly the development of hemoglobin, and probably the seat of the disease is referred correctly to the blood-forming organs, especially the bone-marrow, but how these changes are brought about has as yet eluded discovery. By some the disease is attributed to *auto-intoxication* from the stomach and the intestines, or from the generative organs (ovaries). *Gastroptosis* also has been made responsible for the condition through its injurious influence upon the nervous system, and thus, in turn, upon the generation of blood.

Diagnosis.—The recognition of chlorosis is, as a rule, unattended with serious difficulty. The patients are generally young girls, whose blood exhibits a deficiency in hemoglobin, and whose symptoms generally disappear quickly upon the administration of iron. In the presence of marked gastralgia a suspicion of **round ulcer of the stomach,** with secondary anemia, may be aroused. Care should be observed also not to mistake chlorosis for **latent pulmonary tuberculosis** and anemia. The possibility that an anemic condition may not depend upon chlorosis should always be borne in mind if improvement does not rapidly take place after the employment of preparations of iron, although iron is well borne by the patient.

Treatment.—In the treatment of chlorosis an effort should first be made to remove all **injurious influences** to which the patient has been exposed. The patient should secure as much **rest** for as long a time as possible, and, if the symptoms are severe, even in bed. Long and fatiguing walks are emphatically to be interdicted, although sitting and lying in a hammock in the open air may be permitted. The patient should be advised to practise **cold frictions** morning and evening, a coarse towel being dipped in water at room-temperature and expressed, the trunk and the extremities rubbed for a few seconds, and then with a dry towel. The patient should then remain in bed for fifteen minutes in the morning, and in the evening should at once get into bed after the rubbing, in order to avoid any ill effect from exposure to cold. Frequently I have **sodium chlorid** added to the water, about 50.0 : 2000. The **food** should be easily digestible, and consist principally of milk, eggs, meat, and green vegetables. The belief that chlorosis can be cured by means of a vegetable diet alone, and the use of spinach and cole-slaw, is a serious mistake that will be made only by such physicians as have acquired their experience in therapeutics from the treatment of animals, and in the laboratory, and who are not accustomed to the treatment of sick human beings. Among drugs **preparations of iron** properly play the most important *rôle*.

Personally, I prescribe almost unexceptionally, with admirable results, *Blaud's pills:*

℞ Ferric sulphate,
Potassium carbonate, each, 15.0 (225 grains);
Tragacanth, sufficient to make 100 pills.
Dose: Three pills thrice daily after meals.

Well-to-do patients may prefer to visit the **iron-springs** during the summer, among which Driburg, Pyrmont, Cudowa, Steben, and St. Moritz may be mentioned. **Artificial iron-waters** also have been prepared and have been recommended for domestic use. With all forms of iron-treatment for chlorosis it is important to continue them for a considerable length of time, and to repeat them frequently. Of Blaud's pills I have at least three, and, frequently, six portions taken successively. The results of treatment should be followed by weekly determinations of the hemoglobin-percentage. It but rarely happens that chlorotic patients do not tolerate preparations of iron, particularly Blaud's pills. Under such circumstance **sulphur** has been recommended, although my personal experience with this remedy has not been especially favorable, as either the patients tolerated the sulphur even less well than the iron, or no appreciable influence upon the chlorosis was observed:

℞ Depurated sulphur, 10.0 (2½ drams);
Sugar of milk, 20.0 (5 ").—M.
Dose: 10 grains thrice daily.

I have had no personal experience with the effects of *repeated bleeding* and *courses of sweating,* which have recently been advised from several sources. *Tablets of ovarian extract* also have been recommended of late.

PURPURA.

Purpura signifies merely *hemorrhage into the skin,* although the following description will refer only to those varieties of purpura in which the cutaneous hemorrhage represents the principal symptom of the disease and possesses a certain independence. It is customary to distinguish three varieties of purpura, and to designate them simple purpura, rheumatic purpura or rheumatic peliosis, and hemorrhagic purpura or morbus maculosus Werlhofii, accordingly as the condition is one only of cutaneous hemorrhages (simple purpura), or of cutaneous hemorrhages and articular involvement (rheumatic purpura), or of hemorrhage into the skin, the mucous membranes, and the viscera (hemorrhagic purpura). While a common etiology is indicative of an intimate relationship between the several varieties of purpura, clinical experience also shows that they not rarely pass over one into the other, so that the differentiation is not practicable in all instances. Nothing of a trustworthy nature is known with regard to the *pathogenesis of purpura.* A *bacterial origin* is assumed by many, but in addition a *toxic*, and perhaps also an *autotoxic, mode of origin* must certainly be taken into consideration.

Etiology.—Purpura not rarely develops without demonstrable cause—*cryptogenetic purpura.* In some cases **debilitating disease** has preceded, as, for instance, typhoid fever, pulmonary tuberculosis, carcinoma or chronic diarrhea. Occasionally the disease develops in the course of **infectious diseases,** among which articular rheumatism, pharyngeal diphtheria, dysentery, pneumonia, influenza, syphilis, and gonorrhea may be mentioned. **Intoxications** also are at times followed by purpura (poisoning with phosphorus, arsenic, potassium iodid, mercury, inhalation of sewer-gas). At times **auto-intoxication** appears to be operative, as purpura has been observed after the ingestion of asparagus and decomposing meat. Some cases of purpura are dependent upon **vasomotor disturbances**; for instance, premenstrual and menstrual purpura and the purpura that follows fright. It has not been demonstrated with certainty whether *exposure to cold, excessive physical exertion,* and *emotional disturbances* exert an etiologic influence. *Anemic* and *delicate persons* exhibit a greater predisposition to the disease. The disease occurs more frequently in *adults* than in children.

Symptoms, Diagnosis, and Prognosis.—For purposes of comprehensive description we shall in the following adhere to the schematic classification of purpura into three varieties.

Simple Purpura.—In cases of simple purpura *hemorrhages* occur into the skin. Generally these are inconsiderable in *size*, and only exceptionally exceed that of a pinhead. Such small cutaneous hemorrhages are designated petechiæ. Here and there extravasations have become confluent. Independent larger hemorrhages also occur, which are designated ecchymoses, ecchymomata, and vibices, accordingly as they are flat, nodular, or linear. The *number of hemorrhages* is susceptible of indefinite variation. Occasionally the skin appears as if strewn with hemorrhages closely packed together, while in other patients they are present in smaller number and at greater intervals from one another. Recent hemorrhages exhibit a blood-red *color*, which is not changed by pressure; while older hemorrhages acquire a brownish, bluish-green, or yellow color, in consequence of changes in the hemoglobin contained within the extravasated red blood-corpuscles, and finally disappear wholly. Often a small area of skin surrounding the individual hemorrhage is greenish or yellowish in color, obviously in consequence of imbibition of hemoglobin. It may also happen that some hemorrhages give rise to papular, less commonly to vesicular, elevation of the skin. Occasionally slight cutaneous edema develops. Cutaneous hemorrhage occurs earliest and most constantly upon the *legs.* The upper extremities, and particularly the trunk, often remain exempt. The patients frequently are wholly free from *symptoms*, so that they may discover the cutaneous hemorrhage rather accidentally, or they become conscious of a sense of tension, prickling, even burning in the skin. The patients are generally *pallid*. Often, but not always, diminution in the number of red *blood-corpuscles* and in the percentage of *hemoglobin* is demonstrable. The blood is said also to be deficient in blood-plates. If the patient remains in bed, the cutaneous hemorrhages not rarely disappear quickly, but frequently they reappear for weeks as soon as the patient leaves his bed and stands or walks for any length of time. The disorder is unattended with danger so long as it is uncomplicated. On the other hand, the situation may become grave if internal hemorrhages are superadded or if the disease is transformed into hemorrhagic purpura. Death has in several instances been observed to occur within a short time from intestinal hemorrhage, with symptoms of peritonitis—so-called *fulminant purpura*.

Rheumatic Purpura.—In cases of rheumatic purpura or peliosis the attention of the patients is often first attracted to their disease by *articular pains*. The knee-joints and the ankle-joints are involved with especial frequency, but other joints also are often involved. The painful joints are slightly swollen and exceedingly tender on pressure and passive movement. In a short time, or preceding the articular pains, generally small *cutaneous hemorrhages* occur, which occasionally are particularly numerous in the neighborhood of the painful joints. *Pallor* of the skin and the mucous membranes of rapid development often develops, and diminution in the number of red blood-corpuscles and in the percentage of hemoglobin can be demonstrated. The *bodily temperature* not rarely exhibits slight and irregular elevation. Often a condition of extreme *mental depression* is present. The *duration* of the disease occasionally extends over several weeks, and not rarely numerous remissions and exacerbations in the symptoms occur, the latter principally in consequence of getting out of bed too early, and of standing and walking for considerable lengths of time. *Complications* (endocarditis, pericarditis, stiffness of the joints) occur but rarely, and the disease usually terminates in recovery.

Hemorrhagic Purpura (Morbus Maculosus Werlhofii).—Of all the varieties of purpura the hemorrhagic form is the most serious; nevertheless a fatal issue in consequence even of it is of rare occurrence. At times the disorder sets in with *cutaneous hemorrhages*, to which *hemorrhage into the mucous membranes* becomes superadded, or the reverse order of development takes place. Hemorrhages into the mucous membranes are especially dangerous in consequence of their abundance, difficulty of control, and frequency of recurrence. Occasionally most obstinate and alarming hemorrhages from the gums take place, or there may be bleeding from the nose, hematemesis, enterorrhagia, hemoptysis, or metrorrhagia. Hematuria and symptoms of acute nephritis are often encountered. These especially are not rarely extremely obstinate, and at times persist for more than a year. Frequently hemorrhages into the conjunctiva and the retina occur. Cerebral hemorrhage is of especial gravity, occasionally terminating fatally within a few hours. The disease is often unattended with fever, although irregular febrile movement not rarely takes place. The patients generally are pale, and examination of the blood discloses diminution in the number of red corpuscles and in the percentage of hemoglobin.

The *course* of the disease may be acute, subacute, or chronic. A short time ago I examined a farmer who, five days previously, had been seized with profuse bleeding from the gums. For two days there had been isolated hemorrhages into the skin. On the fifth day of the disease consciousness was suddenly lost, and right-sided hemiparesis appeared. Death occurred in the course of twelve hours. Profuse intestinal hemorrhage, with symptoms of peritonitis, is frequently followed by speedy death.

Among the *sequelæ*, chronic, frequently chronic-hemorrhagic, *nephritis*, *cerebral paralysis*, and *diabetes mellitus* may be mentioned, the last probably in consequence of hemorrhage into the medulla oblongata. Occasionally *multiple sarcomatosis* is said to develop.

Anatomic Alterations.—Opportunities for post-mortem examination in cases of purpura are afforded but rarely, and therefore but little is known with regard to the anatomic alterations of the disease. In addition to the skin and the mucous membranes hemorrhages into internal viscera are not rarely encountered, resulting in part from rupture of vessels, in part through diapedesis. Thrombi are often observed, also, in the small blood-vessels on microscopic examination. The intestinal mucous membrane has occasionally been in a state of hemorrhagic infarction.

Treatment.—In all cases of purpura **rest in bed** and a **light diet** should be recommended. Renewed hemorrhages readily occur on arising too early. Among drugs no marked results will be secured from hematinics—preparations of **iron** and **arsenic**. In cases of rheumatic peliosis **salicylic acid** and **sodium salicylate** (1.0—15 grains—every two hours) have been employed for the relief of the articular pains, but not rarely no effect whatever is observed. In the treatment of hemorrhagic purpura Werlhof recommended **cinchona-bark** and **sulphuric acid**, but these also usually prove ineffective:

℞ Decoction of cinchona-bark,	10.0 : 180;
Dilute sulphuric acid,	5.0 (75 minims);
Simple sirup,	15.0 (½ fluidounce).—M.

Dose: **15 c.c. (1 tablespoonful)** every two hours.

SCORBUTUS (SCURVY).

Etiology.—Scorbutus is a *disease of inanition*; that is, it is a result of insufficient nourishment. It is manifested in a tendency to *hemorrhages* and *inflammations*, among the latter of which *scorbutic gingivitis* occurs earliest and most constantly. The dietetic defects that are followed by scorbutus depend upon either **unsuitable composition** or **insufficient amount of the food.** In earlier centuries scorbutus was encountered with especial frequency in sailors—so-called *sea-scurvy*—and the disease was correctly attributed to the use of salted meat (pickled meat) and to a deficiency in fresh water and vegetables. At the present day sea-scurvy, at one time a much-feared disease, has almost wholly disappeared, as ships can readily supply themselves with meat and canned vegetables, steam-navigation has gained superiority and has notably shortened the length of sea-voyages, and large steamers carry their own live-stock for slaughter and are provided with apparatus for the preparation of palatable drinking-water. Scorbutus occurs not rarely as *war-scurvy*, particularly in besieged cities, when food has become scarce and deficiency in fresh meat and vegetables results. Occasionally scorbutus develops in consequence of *failure of the crops* or of *famine*, particularly after failure in the potato-crops, as the use of fresh potatoes is of great influence in the preservation of health. Prison-scurvy is known to occur, and is a result less of insufficiency than of monotony in and unsuitable constitution of the food. Excessive deficiency of fat in the food has in a number of cases given rise to an endemic outbreak of prison-scurvy. At times the food has exhibited no gross defect with regard to its composition, but it is *insufficient with relation to the demands upon physical activity*. It should also not be overlooked that, in addition to the food, other injurious influences favor the occurrence of scorbutus. Among these are **overcrowding of habitations** (prisons, barracks), **deficient ventilation of habitations,** and **insufficiency of clothing.** Thus, scorbutus has occasionally occurred among explorers who by no means suffered from want of food, but who were unsupplied with a sufficiency of clothing, and were therefore compelled to wear wet clothing until this dried upon their body.

Scorbutus may occur in *epidemic, endemic*, or *sporadic distribution*, as may be readily understood from the nature of the etiologic factors. Often no obvious cause is elicitable for sporadic scorbutus, which occasionally develops unexpectedly after wounds or operations. Adults are generally attacked, less commonly children, in accordance with the nature of the injurious influences, although of late a progressively increasing number of cases of scorbutus have been observed in infants nourished too exclusively with substitutes for milk and artificial foods.

Symptoms, Diagnosis, and Prognosis.—The symptoms of scurvy generally do not set in suddenly, but begin with *prodromes* that eventuate in *scorbutic anemia.* The patient feels languid, is tired, and sweats readily on physical exertion, at the sam time suffering from shortness of breath and palpitation of th heart, and becoming from day to day paler and almost earthy gray Among the symptoms of scurvy **scorbutic gingivitis** occurs earliest The gums become reddened, swollen, and bleed readily on th slightest touch. The swelling is often so considerable that th teeth become surrounded in front and behind by thick masses o mucous membrane, which, in some cases, come in contact at th free margin of the teeth, and bury the teeth between them These alterations occur only about the teeth, and not where th teeth are absent. They begin at the free border of the gums, firs at that surrounding the incisor teeth, are especially marked between the free borders of adjacent teeth, and extend toward th base of the teeth. Occasionally the hyperplastic mucous membrane becomes discolored and gangrenous, the teeth become loos and can be extracted from the jaws with the fingers without forc or pain. The rapidity with which the proliferation of the gum returns after it has been removed in any manner is remarkabl The early and almost constant involvement of the gums is attributed to the fact that in the act of mastication the gums especiall are subjected to mechanical irritation, and therefore are mor readily predisposed to inflammation. Far less commonly than i the gums, similar scorbutic alterations occur at other parts of th oropharyngeal mucous membrane. The patients complain principally of **pain in the gums** and in the mouth, which appears especially in chewing; a **disagreeable odor emanates from the mouth** and they suffer from **salivation** or **ptyalism,** which results fron reflex irritation from the points of inflammation. Not rarely th discharge of saliva is so excessive that the patients are compelle to keep a vessel or a handkerchief constantly before the mouth t receive the fluid. The escaping saliva is frequently admixed wit blood and particles of food, and has an offensive and pungen odor.

Among the **hemorrhages** that are peculiar to scorbutus, *extravasations of blood beneath the skin* occur with especial frequenc These are at times punctate (petechial); but at other times the constitute extensive ecchymoses, or ecchymomata. *Hemorrhag into the muscles* and *beneath the periosteum,* as well as into th *joints,* are not rare occurrences. Such hemorrhages often caus great pain, and interfere with or prevent the functional activit of the affected parts. Occasionally they give rise to inflammator and necrotic softening of the skin, the muscles, or the periosteum with rupture externally and the formation of fistulæ of lon standing. Should cicatrization subsequently occur, shortening o the muscles or adhesions to surrounding structures will readil take place, almost always resulting in deformities and functional disturbances. Hemorrhages into joints not rarely give rise to ankylosis. Occasionally *hemorrhages* take place *from internal viscera,* and these may threaten life by their abundance, resistance to control, and frequent repetition. Epistaxis occurs with especial frequency, but there may also be hematemesis, hemoptysis, enterorrhagia, and bleeding from the female genitalia. Occasionally the *eye* is the seat of hemorrhage. Hemorrhage into the *brain* at times causes death within a short time amid the symptoms of an apoplectic seizure.

Among the *inflammatory alterations* of scurvy scorbutic pleuritis and pericarditis especially are to be mentioned. These are generally hemorrhagic in nature, often representing a hemorrhage rather than inflammation, and are often attended with the danger of death from hemorrhage in consequence of the amount of blood that collects in the serous cavities. Scorbutus is often unattended with *alteration in the bodily temperature,* although irregular febrile movement not rarely occurs. On *examination of the blood* the number of erythrocytes and the percentage of hemoglobin will be found diminished. The *course of the disease* may be acute, subacute, or chronic. Its duration varies accordingly as it is possible to place the patient speedily under more favorable conditions of life. Upon this also the *prognosis* naturally depends essentially. The *recognition of scorbutus* is generally easy. Important diagnostic significance is to be attached especially to the lesions of the gums.[1]

Anatomic Alterations.—The bodies of patients dead of scorbutus are conspicuous for their *emaciation* and *pallor.* Hemorrhages are encountered not only upon the skin and the mucous membranes, but also in many internal organs. Anemic fatty degeneration is often observed. The gums exhibit dilatation and tortuosity of the smaller blood-vessels, and swelling and detachment of the endothelium.

Opinions as to the *nature of scorbutus* are divided. Of late the disorder has been looked upon as an *infectious disease,* but neither the bacterial findings nor the inoculation-experiments upon animals will withstand serious criticism. The unmistakable dependence of the disease upon dietetic errors appears to be indicative of a *toxic etiology.* In this connection *overloading of the blood with sodium chlorid* is considered the actual cause of the disorder by some, who emphasize the fact that scorbutus occurs after excessive ingestion of salted meat, and that alterations resembling those of scorbutus can be induced in frogs by means of infusions of sodium chlorid. Others attribute scorbutus to a *deficiency of potassium in the blood,* and this is ascribed to a lack of fresh vegetables. Possibly both conditions are capable of giving rise to scorbutus.

Treatment.—Prophylactic measures are of great importance in the prevention of scorbutus. In what these shall consist is clear from what has been said with regard to the etiology. Sailors, explorers, and those in besieged cities should supply themselves with an abundance of fresh meat, vegetables, good drinking-water, and clothing. In closed institutions provision should be made for an adequate amount of food containing considerable fat and sufficiently varied, together with systematic ventilation of the rooms. Increased demands upon the physical powers require also increased administration of food, and the like. Scorbutic individuals will do well to leave their homes, and enter a well-equipped hospital or go to a place in the country. Here the principal importance is first to be attached to the diet. The patient should drink milk in abundance, and receive besides fresh meat, fresh vegetables, fresh fruit, butter, and beer. The use of cruciferæ (lettuce, dandelion, radishes, horse-radish, cabbage) especially has been recommended. If thirst be marked, lemonade should be given freely. Among internal remedies **salts of potassium** have been recommended. We have generally combined these with preparations of iron; for instance:

---

[1] In children scurvy often simulates rheumatism, but the diagnosis is easy if the possibility of the former be borne in mind.—A. A. E.

℞ Iron lactate,		
Potassium nitrate,	each,	10.0 (2½ drams);
Arsenous acid,		0.1 (1½ grains);
Powdered althea-root, sufficient to make 100 pills.		
Dose: Two pills thrice daily.		

The patient should be a good deal in the **fresh air**, but at first rather in the recumbent posture than walking about, in order to avoid fatigue and hemorrhage. For the relief of the *gingivitis* astringents and deodorant mouth-washes may be employed, among which I would give first place to aluminum acetate:

℞ Solution of aluminum acetate, 5.0 : 200.
Dose: One tablespoonful to a cup of hot water, for rinsing the mouth after each meal.

Excessive hyperplasia of the gums must occasionally be removed by cauterization with **silver nitrate** or by the **knife**. Other scorbutic symptoms should be treated in the usual manner.[1]

The designation *Barlow's disease* has been applied to a disorder of childhood that occurs particularly in infants in the second half of the second year of life, and represents a combination of scurvy and rachitis. The bones are soft and yielding, and in places [illegible] painful enlargements resulting from subperiosteal hemorrhage. Occasionally epiphyseal separation occurs. If hemorrhage takes place between the diaphysis and the epiphysis, the joints remain [illegible] occurs in consequence of hemorrhage. The [illegible] with good milk, scraped meat and [illegible] particularly mashed potatoes or mashed carrots [illegible].

---

[1] When scurvy develops in infants [illegible] milk be given, together with [illegible].

BLEEDERS' DISEASE (HEMOPHILIA).

Symptoms, Diagnosis, and Prognosis.—The principal manifestation of bleeders' disease consists in the fact that even slight **traumatic hemorrhage** can be controlled with difficulty, if at all, and may threaten life; or that external or internal *hemorrhage* occurs *spontaneously* from time to time, and which likewise may become alarming from the difficulty in controlling it. Death often occurs in the *newborn* in consequence of hemophilia, as a result of hemorrhage from the umbilical wound. Occasionally the disorder is latent for a considerable period of time, and only becomes manifest in the form of uncontrollable hemorrhage in connection with an accidental wound. A pin-prick, puncture of the gums, the extraction of teeth, a leech-wound, laceration of the hymen in the act of defloration, may be followed by fatal hemorrhage. Occasionally surgeons have recognized hemophilia from the fact that death has occurred as a result of hemorrhage from harmless incisions in patients subjected to operation. In some patients, however, the disorder is revealed by certain manifestations that are particularly significant if the patients are members of bleeders' families. **Pains in the muscles and the joints** occur with especial frequency; they are associated with swelling, and are dependent upon hemorrhage. As a result the joints occasionally become deformed and ankylotic. The clinical picture becomes more distinct if at times **spontaneous hemorrhages** occur externally, which are often preceded by palpitation of the heart, shortness of breath, a sense of fear, a feeling of fulness and beating in the head as prodromes, manifestations that disappear as soon as the blood has made its escape. **Epistaxis** occurs most frequently, but also extensive hemorrhages often take place into the **skin,** the **muscles,** and the **joints.** **Hemorrhages into the stomach and the intestines** are less common, while *renal hemorrhage* is more frequent. At times repeated spontaneous renal hemorrhage constitutes the only symptom of hemophilia. In some patients the hemorrhages recur with a certain degree of regularity. If the hemorrhage takes place externally, the blood will be seen to escape uninterruptedly from a wound, without the discovery of a spurting or even of a bleeding vessel being possible. The designation *capillary hemorrhage* is therefore also employed. Naturally, the patients appear pale after copious loss of blood, but the rapidity with which recovery ensues is generally remarkable. *Examination of the blood* has thus far disclosed no peculiar condition, although a slight tendency to coagulation, and marked softness of such clots as form may be recognized. Perhaps these features depend upon alterations in the colorless blood-corpuscles, whose number is often said to be diminished—**hypoleukocytosis**—and also in the blood-plates.

Hemophilia is an incurable disease, which exposes the patient to the danger of death from hemorrhage, and the *prognosis* is therefore serious. Occasionally, it is true, the hemophilic symptoms become less prominent with advancing years, and, in rare instances, they eventually disappear. The *recognition* is not always easy, and at times the diagnosis is made only after death has resulted from hemorrhage.

Anatomic Alterations.—The organs of patients with hemophilia dead from hemorrhage naturally exhibit signs of **anemia,** and are not rarely the seat of hemorrhages and of anemic fatty degeneration. Distinctive lesions are not known. The heart and the blood-vessels have in some instances been characterized by deficient development (hypoplasia). Irregular formation and deficiencies, swelling, fatty degeneration and detachment of the endothelium have also been observed in the smaller blood-vessels.

Nothing is known as to the *nature of hemophilia.* The tendency to spontaneous hemorrhage is indicative of abnormally increased *blood-formation.* Besides, deficient *constitution of the blood* should be taken into consideration, interfering with the coagulation of the blood. Where the seat of the disease is to be looked for, and wherein the active injurious influence consists, are unknown.

Etiology.—Hemophilia is, in most cases, an *hereditary disease;* particularly in the lonely valleys of Grisons in Switzerland, and in the Rhine countries families are known in which the disorder has been transmitted by inheritance for centuries. It is noteworthy that the males in families of bleeders do not transmit the disease to their descendants if their wives are members of healthy families; while, on the contrary, women in hemophilic families generally remain free from hemophilia, although they transmit the disease to their male descendants, even if the husband is a member of a healthy family. Women, therefore, generally do not suffer from hemophilia, but constitute the medium for its transmission, while the males are bleeders, but do not further transmit the disease. Not rarely some members of a family of bleeders remain free from hemophilia. It may also happen that some generations remain exempt. *Acquired hemophilia* is observed much less commonly. Pulmonary tuberculosis, scrofulosis, rheumatism, and gout in the parents are considered as causative influences, but often no etiologic factor whatever is elicitable. Further, it is not always easy to differentiate acquired and hereditary hemophilia with certainty, for although the development of symptoms of hemophilia after the twentieth year of life is indicative rather of the acquired disease, it may naturally at times occur also in cases of hereditary hemophilia that the first opportunity for traumatic hemorrhage is afforded in later life. Acquired hemophilia may become the source of origin for hereditary hemophilia. Under all circumstances hemophilia is fortunately a *rare* disease, which occurs almost exclusively in the *Indo-Germanic race*, and particularly in *men*.

Treatment.—The most reliable **preventive measure (prophylaxis)** against hereditary hemophilia is the non-marriage of the female members of hemophilic families, and, as a matter of fact, the girls in the hemophilic families of Grisons have agreed by vow to remain single. Measures for the control of existing hemophilia are unknown. Particular importance is to be attached to the **avoidance of all wounds.** Naturally, all dangers are thereby not averted, as life may be threatened also by spontaneous hemorrhage. Among wounds only vaccination appears to be borne by the hemophilic, while circumcision of the prepuce is dangerous. Bleeders should be exempt from military service. If *traumatic* or *spontaneous hemorrhage* occur, it should be controlled according to surgical rules by tamponade, elevation of the member, constriction or ligation of the vessels. Occasionally success will be attained by enlarging the wound with the knife, and instituting surgical treatment. Renal hemorrhage has also been controlled by nephrectomy. Nothing can be accomplished with internal *hemostatics*, *styptics*, or *astringents*. Calcium chlorid (2.0—30 grains—daily) has been reported to exert a favorable influence through its power of increasing the coagulability of the blood. If the anemia is alarming, **physiologic salt-solution** (0.75 per cent.) may be infused into the veins.

PAROXYSMAL HEMOGLOBINURIA.

Symptoms, Diagnosis, and Prognosis.—In cases of paroxysmal hemoglobinuria the patients secrete periodically **bloody urine,** which, however, contains few or no red blood-corpuscles, but hemoglobin in solution; there thus results *hemoglobinuria.* This hemoglobinuria is always preceded by *hemoglobinemia;* that is, solution of red corpuscles in the blood first takes place, and the blood-plasma gets rid through the kidneys of the hemoglobin dissolved in it. Another portion of the hemoglobin is deposited in the spleen and the liver, and causes enlargement of both of these viscera. The occurrence of an attack of hemoglobinuria is often preceded by the operation of certain injurious influences, including especially **exposure to cold** and **excessive physical activity.** In some patients an attack may be induced with certainty by a cold bath or a long walk. The patients also occasionally suffer from attacks only during the winter; whence the name *winter hematuria.* Often the attack begins with **chilliness,** or even with a chill, which is followed by **elevation of temperature** to 40° C. (104° F.) and above. The patient acquires a remarkably pallid, almost **earthy appearance,** while not rarely the skin and the conjunctival covering of the sclera are distinctly, though generally but slightly, icteric. Occasionally **urticaria** is present. The **spleen** and the **liver** become increased in size, and are not rarely tender on pressure. Also, complaint is often made of a sense of tension and of pain in the region of the kidney.

On examination of the *blood* the plasma exhibits a bloody color, and here and there discolored red blood-corpuscles—so-called blood-shadows—are visible. The number of red blood-corpuscles and the percentage of hemoglobin are diminished. The **urine** may possibly contain only a small amount of albumin, but it soon becomes conspicuous from its bloody color, which is not like that of fresh red blood, but is rather reddish brown or almost black from the presence of methemoglobin in considerable amount. If the urine be boiled, a coherent mass of albumin presenting a brownish appearance, from the presence of hemoglobin, will usually be precipitated. On spectroscopic examination of the blood either the absorption-bands of methemoglobin alone are found, of which particularly a band in the red of the spectrum between the Fraunhofer lines C and D is important; or the two bands of oxyhemoglobin in the yellow and the green between the lines D and E appear. On microscopic examination of the urinary sediment granules and filaments of hemoglobin will be found, which here and there lie close together in the form of hemoglobin-casts, and also a small number of red blood-corpuscles, mostly in the form of decolorized discs or shadows. If round cells or epithelial cells from the uriniferous tubules are present in the sediment, these also are often found discolored greenish or brownish by the hemoglobin.

The attack of hemoglobinuria is of variable *duration*, lasting between a few hours and a few days. Slight albuminuria often persists for a short time after the hemoglobinuria has disappeared. *Abortive attacks* also occur, manifested only by paroxysmal albuminuria. The disorder can be readily recognized, but not rarely proves most *obstinate*, although it but seldom terminates fatally, so that the *prognosis* is not unfavorable.

Anatomic Alterations.—Opportunities for post-mortem examination in cases of paroxysmal hemoglobinuria have as yet been exceedingly rare. The kidneys have been found abundantly filled with blood and enlarged, and they contained hemoglobin.

Etiology.—**Syphilis** should be mentioned as a not uncommon cause of paroxysmal hemoglobinuria; also, other **infectious diseases**—malaria, articular rheumatism—are occasionally followed by the disorder. Such other influences as have been mentioned as causative factors cannot be considered as such. Obviously syphilis is capable of diminishing the resisting power of the red blood-corpuscles, for it has been possible to demonstrate that in such patients constriction of a finger and immersion in cold water will cause dissolution of the red blood-corpuscles. The disease is most common in *men*.

Treatment.—During an attack of hemoglobinuria the patient should be kept in **bed** and weak **tea** administered. In order to avert the occurrence of an attack, the patient should guard against exposure to cold, cold baths, and excessive physical activity, especially long walks. The general tendency to the disease will be removed by **mercurial inunctions** and **potassium iodid** if syphilis be the cause of the disorder. *Quinin* is to be recommended when malarial influences are operative.

PART IX.

DISORDERS OF METABOLISM.

OBESITY (POLYSARCIA).

Etiology.—Polysarcia depends upon **abnormal formation and accumulation of fatty tissue** in the body. Two varieties of obesity can be distinguished, namely, the *plethoric* and the *anemic*. Plethoric obesity is the result of *faulty nutrition*. One who indulges in larger amounts of food than are demanded by his physical activity is exposed to the danger of becoming obese. The disorder therefore occurs frequently in persons who *move about but little* and who engage in *little physical exercise*. Obesity often depends upon **faulty constitution of the food,** and such individuals therefore become obese who use carbohydrates (farinaceous food, sugar, alcohol) in excess. In addition, the tendency to obesity is *hereditary* in some families, so that in the members of such families an excessive deposition of fat takes place, even with the observance of an apparently moderate mode of life. Often several of the injurious influences named are jointly operative, and the patient, in spite of hereditary predisposition, eats generously, with a preference for farinaceous and saccharine food, indulges freely in alcoholics, and avoids all possible physical movement and activity.

From what has been said it should not be surprising that certain *periods in life* favor the development of obesity. Infants often exhibit abundant deposition of fat because the milk is rich in sugar and active physical exercise is wanting. Also, however, in advanced age, when it is customary to withdraw gradually from ordinary pursuits, the increased physical inactivity readily results in obesity. Rarely obesity is congenital. **Protracted sleeping** favors the development of obesity, as well as a moist, warm **climate** and **sexual continence.** Eunuchs readily become obese. Obesity likewise develops readily in women at the climacteric period.

Anemic obesity is observed in connection with chlorosis, progressive pernicious anemia, and allied conditions. It occurs also after loss of blood, not rarely after antecedent parturition. Even in cases of carcinoma and pulmonary tuberculosis an excessive deposition of fat not rarely takes place. Under all of these conditions the increased formation of fat depends upon the deficiency of hemoglobin in the blood, resulting in diminished processes of oxidation and combustion in the body. In this category belongs also *toxic obesity*, which is of but slight clinical significance, and, among other factors, develops after the use of arsenic.

Anatomic Alterations.—In cases of obesity the fatty tissue is deposited first in increased amount in such situations as under normal conditions are the seat of fat, and only subsequently does it appear upon unusual portions of the body. The **subcutaneous fatty tissue** acquires a considerable thickness; particularly in the abdominal walls it often forms a layer 10 cm. thick and even more. The **mediastinal fatty tissue** also is greatly increased, and represents a thick, coherent layer of fat. The **great omentum,** the **mesentery,** and the **epiploic appendages of the intestine** represent enormous masses of fat, between which bowel and other abdominal viscera appear in some degree squeezed. The *heart* is surrounded by an enormous fatty capsule, in consequence of increase in the subepicardial fat, and this often penetrates between the muscular layers of the heart. The kidneys likewise appear buried in a massive capsule of fat. Other viscera, particularly the **liver** and the **kidneys,** are enlarged, generally anemic, and infiltrated with fat. Even in the blood an excessive amount of fat has in a number of instances been described—so-called lipemia; but, it is true, all such reports do not appear trustworthy.

Symptoms, Diagnosis, and Prognosis.—Obese individuals attract attention, in the first place, on account of their increased size; the **abdomen** particularly becomes greatly increased in circumference. If the abdominal walls be grasped, a remarkably thick layer of fat can be felt. The umbilicus and the genitalia not rarely appear greatly retracted, while the **breasts** project as heavy cushions of fat. The entire **shape of the body** becomes rather oval, and the **attitude of the body** is so changed that the patients walk almost like a pregnant woman, with the upper part of the body bent backward, with the legs far apart, and with short, stumbling steps, so that they make the impression of a sprawling and bloated individual. The **bodily weight** increases, and not rarely attains extraordinary figures (up to more than one thousand pounds); while, on the other hand, the **specific gravity** of the body diminishes, so that obese persons float readily in water, particularly in salt sea-water.

The **facial expression** of an obese person often exhibits something of fatigue and dulness. The fat cheeks hang loosely downward, the eyelids overladen with fat cause narrowing of the palpebral fissure, and the fatty tissue in the submental region projects downward as a thick cushion of fat—so-called double chin—so that the chin appears retracted. The **nucha** is thickened and shortened, and frequently forms one or several transverse rolls of fat.

Upon the **dorsum of the hand** the masses of fat project like cushions. In plethoric obese individuals the face generally exhibits a markedly red, congested appearance, and dilated and tortuous small blood-vessels can often be recognized beneath the skin. In cases of anemic obesity, however, the pallid skin is frequently striking, and not rarely the color is one of waxy pallor. At the corneoscleral junction a *senile arc* (*gerontoxon*) is not rarely present, resulting from fatty degeneration of corneal corpuscles. Obese individuals present an appearance of *clumsiness* and *indolence*. Stooping is attended with great difficulty and physical exertion is generally avoided, for one reason because it is followed by **distressing sweats.** The obese individual is altogether greatly disposed to sweating. He, therefore, readily suffers from exposure to cold, and complains frequently of **pains in the muscles and joints.** The **sebaceous secretion of the skin** also is increased, and the sweat, therefore, often acquires a fatty character. The skin exhibits a tendency to inflammation, and eczema, intertrigo, and acne readily develop. Frequently **mental indolence** is observed in obese individuals, with the development of a marked aversion to mental activity.

With the rather *general symptoms of obesity* thus far described are not rarely associated **local disturbances.** The *symptoms due to fat heart* become apparent with especial frequency. The patient often complains of palpitation of the heart and irregularity in cardiac action. The sounds of the heart are faint, the cardiac dulness appears increased in extent and marked, and conditions of cardiac asthma and other indications of fat heart, such as have been described on page 30, Vol. I., rapidly arise. Should insufficiency of the cardiac muscle develop, cutaneous edema and other symptoms of stasis will appear. Obese individuals often complain of persistent **dyspnea** and **shortness of breath,** which are explicable from the fact that the mobility of the chest is interfered with, and adequate expansion of the lungs is prevented by the increased amount of fatty tissue in the mediastinum, upon the heart, and in the abdominal cavity. *Fatty liver* would be indicated by increase in the size of the organ, with a sense of pressure in the right hypochondrium, while jaundice never occurs in the presence of the uncomplicated fatty liver. **Gastro-intestinal disturbances** are often present, and are due in part to excessive eating, and in part to impaired peristaltic activity. **Hemorrhoids** frequently develop. **Sexual desire** is frequently diminished in the obese. Not rarely mechanical **impotence** exists, the hyperplastic masses of fat preventing copulation, although a deficiency of spermatozoids in the seminal fluid has also been observed. Obese women often suffer from displacements of the generative organs and from amenorrhea.

The *recognition of obesity* is scarcely attended with difficulty, although it has happened that a pendulous abdomen has been mistaken for ascites, and has been punctured. The *variety of obesity* also can generally be determined without difficulty.

The *prognosis* is not favorable, for even when it is possible to correct the obesity the treatment requires abstinence from numerous pleasurable indulgences, to which many patients are but rarely willing to submit permanently. This is applicable especially to plethoric obesity. The existence of obesity is associated with various dangers. Some patients succumb to progressive *cardiac weakness* and manifestations of stasis. Not rarely death results in obese individuals in consequence of *cerebral hemorrhage,* obviously because cerebral vessels have undergone fatty change and have ruptured. In general, obesity creates a predisposition to vascular alterations, particularly *arteriosclerosis.* Occasionally death results suddenly in consequence of *fat-necrosis* and *hemorrhage into the pancreas.* It should not be overlooked that obesity is not rarely associated with other serious disorders of metabolism or occurs as a prodrome. Among these may be mentioned the formation of *renal* and *biliary calculi, gout,* and *diabetes mellitus.*

Treatment.—For the *relief of plethoric obesity* there is but one remedy of permanent effect, namely, an **intelligent diet** and **mode of life.** The patient should not take more food than is necessary. He should, so far as possible, avoid all fat-forming foods (farinacea, sugar, alcohol), and take as little liquid food as possible. He should indulge in appropriate activity and exertion, and restrict sleep to eight hours. The use of fat is not alone permitted, but is even to be recommended, because it does not produce fat and it lessens the sense of satiety and thirst. Bread, cake, pastry, maccaroni, potatoes, and alcohol are especially to be forbidden. The use of warm baths (35° C.—28° R.—95° F.) twice weekly is to be advised. Walks should be taken in the morning and the afternoon of a total duration of from two to three hours, and the ascent of elevations is commendable for the purpose of abstracting water from the body through copious sweating. The mode of life described requires a certain degree of renunciation and self-control on the part of the patient, which some do not possess at all, while others may persevere for but a short time and soon relapse into their previous habits, when they soon regain the weight lost.

Several *denutrition-cures* have been recommended, of which each is capable of attaining the desired end, confirming the old saying that many roads lead to Rome. The *system of Banting* allows the patient an abundance of lean meat, but forbids as strictly as possible the use of carbohydrates and fat. The *system of Ebstein* likewise restricts the ingestion of carbohydrates, and places stress upon the generous use of fat. In the *system of Oertel,* finally, more carbohydrates and less fat are permitted, and the ingestion of fluids (soups, beverages) especially is restricted.

The mode of life that I prescribe is generally as follows: The patient should retire to bed at 10 P. M. and arise at 6 A. M. At 7 A. M. he should receive a large cup of tea (about 250 c.c.) without sugar, and two rolls (50 grams) generously spread with butter. At 1 o'clock he is given 150 grams of broiled meat or fish, green vegetables, and fresh fruit. No soup at all is given, or not more than 250 c.c., and then meat-broth, with a small amount of bone-marrow. No wine at all is permitted, or a wine-glassful of light domestic wine an hour after a meal. At 3 P. M. a small cup (150 c.c.) of coffee with cream, but without sugar, is given; at 7 P. M. 50 grams of bread with butter and cold meat, particularly fat ham, blood-sausage, liver-sausage, or meat-sausage, and one or two eggs, together with a large cup of tea without sugar.

In the first days of treatment the patients are generally annoyed by distressing thirst, which is best suppressed with a little encouragement, it being especially explained to the patient that many persons drink not from necessity, but from habit. If absolutely necessary, small bits of ice may be permitted to melt slowly in the mouth. Violent courses of denutrition should be avoided, as they may give rise in many patients to palpitation of the heart, vertigo, a sense of fear, and even alarming exhaustion.

Among *drugs* that cause reduction in fat, **potassium iodid** (5.0 : 200; 15 c.c.—one tablespoonful—thrice daily) may be mentioned, although iodin-cachexia is to be feared if it be too long continued. Recently thyroid extracts have been recommended, although in several cases under my observation they have proved disappointing, apart from the fact that occasionally the agent gives rise to palpitation of the heart, vertigo, tremor, even albuminuria and glycosuria, when it must be withheld.

Drinking-cures (Marienbed, Kissingen, Homburg, Tarasp) are attended only with transient benefit, which soon disappears after the conclusion of the treatment.

In cases of *anemic obesity* all fat-forming foods should likewise be avoided, and, in addition, preparations of iron should be prescribed, in order to relieve the anemia.

GOUT.

Etiology.—Gout is known also as the *uric-acid diathesis*, a designation that is intended to indicate that the abnormal metabolism is attended with the formation of excessive amounts of uric acid, which predisposes the body to inflammatory processes in various organs. A distinction should be made between *hereditary* and *acquired gout*. *Hereditary gout* may occur even in such individuals as pursue a regular and healthful mode of life. It is true, it will develop the more certainly if certain other injurious influences have been operative that are alone sufficient to induce gout in persons without any hereditary predisposition. Among these are **improper food** and **toxic influences**. *Dietetic errors* include especially over-eating and the ingestion of excessive amounts of carbohydrates and alcohol. The occurrence of gout under such circumstances is further favored by insufficient physical activity and exercise; as, however, the same conditions also favor the development of obesity, it can be understood that gouty patients are frequently obese. This variety of gout may be designated also *patrician's gout.* Clinical experience teaches, however, that gout may be a result also of privation and deficiency of food; and *plebeian's gout* may thus be recognized in contradistinction from patrician's gout. The *toxic form* of gout includes that due to *lead-poisoning,* which is observed in laborers and artisans who are exposed for a long time to the influence of lead (painters, potters, printers). Gout occurs only exceptionally in childhood, and it generally does not appear before the *thirtieth year of life.* The fact that *men* are attacked more frequently than women is due, in part, to the nature of the etiologic factors. Gout occurs more frequently in some *countries* than in others; thus the disease is rare in Switzerland, while it is common in England and in Holland.

Symptoms, Diagnosis, and Prognosis.—Gout is generally a chronic disease, with a marked tendency to recurrence. Most frequently it begins as *acute articular gout,* then gradually acquires the characteristics of *chronic articular gout,* and finally terminates as *visceral gout.* Nevertheless, variations from this typical course not rarely occur; articular gout particularly may be wanting, and only visceral gout be present—conditions that are often difficult of recognition. *Acute articular gout* is often preceded by **premonitory manifestations** or **prodromes.** The patient complains of a coated tongue, a bad taste, acid eructation, a sense of fulness, a feeling of pressure in the head, and the like. The attack of acute articular gout with preference first involves the metacarpophalangeal joint of one great toe, most frequently the left, and here gives rise to the symptoms of podagra. An individual who has retired to bed wholly free from pain may be awakened from sleep shortly after midnight by frightful pain in the joint of the big toe. The pain is described as burning, boring, cutting, crushing, as if the great toe were grasped within a vise and were being slowly crushed. The affected joint is swollen, the overlying skin reddened and shining, and not rarely dilated and tortuous cutaneous vessels can be seen coursing toward the periphery, and, on palpation, the skin feels unusually hot. The slightest pressure and the gentlest movement of the joint induce the most intense pain. Only toward morning does the pain recede somewhat, generally after slight sweating has appeared upon the skin. In the succeeding nights the conditions described may be repeated, but they gradually become less marked, and toward the end of the week they remain entirely absent. The patient has now passed through his attack of acute articular gout. The joint remains stiff and its mobility will be impaired for a few days, but the swelling then wholly subsides and the previous mobility is restored. Not rarely desquamation of the skin takes place.

Although an attack of acute articular gout occasionally occurs without appreciable cause, it frequently follows **indiscretions in** diet, in drinking, and sexual indulgence. Occasionally the patients are so alarmed by the attack of gout that they are induced to live more carefully in the future, in order to avoid recurrence of the disease. It is naturally the rule that such care is observed for only a short time, when the former excesses are again indulged in, and thus renewed attacks of acute gout occur from time to time. At first these attacks also often involve only the joints of the great toe, and complete restoration of the joint takes place. Gradually, however, other joints become involved in inflammation and swelling, the subsidence of the inflammatory alterations occurs more and more slowly, and particularly more and more incompletely. Above all, indurated enlargements and excrescences remain in the joints, consisting of uric acid and acid sodium urate, and which are designated **gouty nodules or arthritic tophi.** The articular surfaces become more and more greatly deformed, grate on active and passive movement, and the joints become stiff and often wholly immobile. The deformity of the joints is not rarely quite considerable, and the fingers may as a result acquire an appearance that has been compared with that of parsnip-root. It may also happen that the fingers become displaced with especial frequency toward the ulnar aspect, and superimposed upon one another like shingles on a roof, as in cases of deforming arthritis, and are rigid. In some cases the alterations described, and which constitute the picture of *chronic articular gout*, remain confined to a single joint, while in other instances a large number of joints are involved. It may also be observed that in some patients the articular alterations vary and change in place, while in others they remain confined to the same joint, so that a distinction is made between *vague* and *fixed articular gout*.

The clinical picture of chronic gout includes also the development of *gouty nodules* in other portions of the body. Thus, *gout of the bursæ* is manifested by swelling of these structures, which are gradually transformed into firm nodular deposits of uric acid and urates, and which form enlargements of stony hardness, as large as a fist, particularly at the elbow or over the patella. *Gout of the tendon-sheaths* is attended with circumscribed enlargement, and the subsequent formation of gouty nodules in various tendons, and with especial frequency in the extensor tendons of the forearm. *Gout of the cartilages* occurs with especial frequency in those of the ear, and gives rise in this situation to the presence at the free margin of the auricle of whitish nodules averaging the size of a pin-head, which are often surrounded by dilated blood-vessels. At times gouty nodules have also been observed upon the cartilages of the larynx, and have been recognized with the laryngoscope. They occur, also, upon the palpebral and the nasal cartilages. Reference may be made also to gouty nodules beneath the *periosteum* and in the *penis,* as well as in the *skin,* particularly that of the face. *Gouty ulcers* occasionally result from gouty nodules. Under such conditions the overlying skin becomes reddened and inflamed, undergoes destruction, and chalky-white masses of stony hardness come into view, which, upon microscopic examination, are found to consist of fine needles. The latter yield the murexid-test (heating with nitric acid and appearance of a reddish discoloration on addition of ammonia, and of a bluish discoloration on addition of potassium hydroxid) and thus reveal their uric-acid composition. The surface of the ulcers exhibits a slight tendency to suppuration and granulation, and to cicatrization. Small gouty nodules, as, for instance, those in the auricles, are sometimes wholly exfoliated, and leave a small permanent depression.

Gout of the internal viscera is a serious disease, and indicates that the body, overladen with uric acid, is greatly predisposed to inflammatory processes of all kind. The kidneys and the heart are involved with particular frequency. The **kidneys** not rarely become the seat of chronic interstitial inflammation, with all its consequences (light, abundant urine of diminished specific gravity, small amount of albumin and sediment, hypertrophy of the left side of the heart, heaving apex-beat, wiry pulse), and in some gouty patients death results from uremia or cerebral hemorrhage. With reference to the **heart,** insufficiency of the myocardium and angina pectoris readily occur, but also endocarditis, pericarditis, and fibroid degeneration of the myocardium are not uncommon conditions. Arteriosclerosis and aneurysm, likewise, are often due to gout. In addition to the foregoing, inflammatory processes have also been observed in many other organs, as, for instance, pneumonia, pleurisy, inflammation of the bronchial, tracheal, and laryngeal mucous membrane, pharyngitis, rhinitis, ophthalmitis, interstitial connective-tissue hyperplasia in the liver, gastritis, enteritis, even inflammatory discharges from the urethra, etc.

If, in addition to the inflammatory processes in question, gouty changes in joints and gouty nodules are also present, the etiologic diagnosis is easy; but otherwise it may be impossible, or can be made if saturation of the body with uric acid is demonstrable. To this end the application of a cantharidal plaster and examination for uric acid of the contents of the blister that develops have been advised. For this purpose the *thread-test of Garrod* may be employed, the contents of a cantharidal blister being collected in a watch-glass, five drops of acetic acid added, a coarse cotton thread introduced, and from twelve to twenty-four hours permitted to elapse. Should uric acid in increased amount be present in the contents of the blister, colorless four-sided plates of uric acid will have been deposited upon the cotton thread, and whose identity can be readily demonstrated chemically by means of the murexid-test already referred to. The thread-test described will also be of value if gouty nodules are not present in a case of gout, and the question arises whether the condition be one of gouty or of deforming arthritis.

Among the *complications* of gout the frequent association with *obesity* has already been referred to. In addition, *diabetes mellitus* and the formation of *renal* and *biliary calculi* may further be mentioned. Long continuance of the disease occasionally gives rise to *amyloid degeneration*. Gout is, under all circumstances, a serious disorder, which recurs from time to time with increased intensity, even in spite of a careful mode of life, to which naturally all gouty patients will not submit, and in addition life may be threatened in most varied ways. Accordingly, the *prognosis* of gout is grave, and particularly so with regard to cardiac and renal gout. Nevertheless, gouty patients not rarely attain advanced age, although frequently rendered helpless invalids, harassed by constant pain, and dependent upon the assistance of others.

Anatomic Alterations.—The peculiar anatomic characteristic of gout consists in the **deposition of urates** in the most varied viscera. When this is absent it is impossible to differentiate with certainty between inflammatory processes of gouty and non-gouty origin. In accordance with the clinical manifestations deposits of urates are encountered most frequently in the **joints**, where they are at first appreciable as small chalky-white dots and lines beneath the surface of the articular cartilage, but they progressively increase in size, coalesce into larger coherent surfaces, and give rise to destruction and deformity of the articular surfaces. The **articular capsule**, the **bursa**, the **tendons**, the **periosteum**, the **auricle**, and other structures are also the seat of more or less extensive deposition of urates. The **heart** frequently exhibits arteriosclerotic, often calcareous thickening, as does also the intima of the **aorta**; and it has been possible by means of the murexid-test to demonstrate the presence of uric acid in these structures. The myocardium not rarely contains fibroid areas, and even it may be the seat of uratic deposits. The **kidneys** often appear contracted. They are diminished in size; the capsule is frequently adherent to the cortex and the surface often exhibits a multinodular appearance. On section the cortex is diminished; at the same time the consistency of the kidneys is increased. That such contracted kidneys are *gouty* organs can be recognized only from the presence of whitish deposits of urates, which are situated in part in the cortex, in part in parallel lines in the medullary structure, and, on microscopic examination, are found partly within the uriniferous tubules, and partly within the interstitial connective tissue. The **liver** is often in a condition of chronic interstitial hyperplasia of the connective tissue, or cirrhosis.

Much discussion has been waged with regard to the *nature of gout*, and even at the present time there is no unanimity of opinion on this subject. It may be considered as established that the condition is attended with *abnormally increased production of uric acid*, as uric acid can be demonstrated in the blood and in the contents of cantharidal blisters in cases of gout, and not in individuals free from gout. While formerly the increased formation of uric acid was attributed to diminished oxidation-processes in the albuminous materials of the food, the uric acid and its chemically related alloxuric bodies are at present thought to be derived from the *nuclein-bodies* of the cell-nuclei, and it has been contended that by means of Ehrlich's triacid solution peculiar granules have been found in the colorless blood-corpuscles in cases of gout, which are looked upon as the material out of which uric acid is formed. Nevertheless, it should not be imagined that the formation of uric acid is confined solely to the blood. Obviously, it takes place in the most varied viscera. That a body saturated with uric acid is predisposed to inflammatory processes of all kind should not appear remarkable. That particularly articular gout occurs with especial frequency and constancy is probably dependent upon the fact that the joints are peculiarly exposed to traumatism and pressure, that of the great toe in the erect posture, and that joints become more readily susceptible to injurious toxic and bacterial influences the more severe the mechanical injuries to which they have been subjected. The periodic occurrence of articular gout, and of gouty manifestations generally, would yet require explanation. This might be dependent upon temporary increase in the formation of uric acid, or upon periodic diminution in the elimination of uric acid through the kidneys. Nevertheless, cases of gout occur, and such a one was recently under my care, in which the elimination of uric acid through the urine was diminished at the time of an attack of acute articular gout. This does not, however, take place unexceptionally, and under such conditions it must be assumed either that a diminution in the amount of uric acid had taken place some time in advance of the attack of articular gout, and which had been compensated for at the time of the attack, or that in these cases there occurs a temporary increase in the formation of uric acid. The *occurrence of gouty deposits* has been explained by assuming that local stasis in the movement of the fluids of the body gives rise to stagnation of the uric acid in various tissues, where the uric acid causes necrosis. The dead tissue acquires an acid reaction, and thereby causes precipitation of the soluble neutral sodium urate in the form of acid sodium urate. Recently the correctness of this explanation has been questioned.

Treatment.—**Causal treatment** should be directed to regulation of the *diet* and the *mode of life*, and gluttons, drunkards, and indolent persons should observe all the regulations laid down on pp. 278, 279 for the relief of obesity. The gouty patient should especially avoid food rich in nuclein (thymus, liver, kidney, brain, game, salt meat, cheese). In cases of plebeian gout generous indulgence in meat should be advised, and in cases of gout due to lead, potassium iodid (5.0 : 200; 15 c.c.—one tablespoonful—thrice daily), and sulphur-baths (potassium sulphid 150.0 for a full bath at a temperature of 35° C.—28° R.—95° F.) should be prescribed, and the patient be permanently removed from the influence of lead. Drugs have been recommended as **specific remedies** that are capable of dissolving uric acid and its salts, although it is highly questionable whether these are capable of exerting any effect in the great dilution in which they are introduced into the body. In this group belong the *lithium-salts* (lithium carbonate, lithium salicylate, lithium chlorate), *artificial lithia-water* and *lithium-springs* (Assmanshausen, Baden-Baden, Dürkheim, Elster, Homburg, Kissingen, and Obersalzbrunn). The lithium-salts are given in doses of from 0.3 (4½ grains) to 0.5 (7½ grains) thrice daily, but gastric derangement should be looked for, as this may readily occur from the use of these salts. Among the solvents of uric acid are also *lysidin, lycetol, piperazin, uricedin,* and *urotropin,* all of which may be administered in doses of 1.0 (15 grains) thrice daily. Among the actual specifics for gout *colchicum* must yet be mentioned, constituting the principal ingredient of a number of secret remedies, among others also the widely known Liqueur Laville. Care should be taken to avoid excessive doses of colchicum, as they may readily induce alarming cardiac weakness:

℞ Tincture of colchicum,
Tincture of aconite, each, 5.0 (75 minims).—M.
Dose: 15 drops thrice daily after meals.

The persistent use of *alkaline waters* should be recommended to gouty patients, particularly those of Fachingen, Kronenquelle, Obersalzbrunn, Offenbach, Kaiser Friedrichsquelle, Wiesbaden Kochbrunnen, Evian, and Vals. Recently the generous use of oranges or lemons—the so-called **orange-cure** or **lemon-cure**—has been recommended, although the results are highly doubtful.

For the *relief of acute articular gout* **rest** and **elevation of the part** are especially to be recommended. Little success will, as a rule, be secured from the administration of *salicylic acid, sodium salicylate, lithium salicylate, antipyrin, phenacetin, salophen, salipyrin;* nevertheless they should be employed (1.0—15 grains—every two or three hours). In addition, the affected joint should be massaged morning and night with **salicylated ointment** and be wrapped in **salicylated cotton**:

℞ Salicylic acid, 5.0 (75 grains);
Wool-fat,
Lard, each, 25.0 (¾ ounce).—M.
Use by inunction thrice daily.

For the relief of *chronic articular gout* **saline, sulphurous,** and **indifferent thermal baths** have been recommended, while *visceral gout* is to be treated in the same way as corresponding disturbances due to other than gouty influences.

DIABETES MELLITUS.

Etiology.—Diabetes mellitus results from excessive accumulation of glucose in the blood—*hyperglycemia* (instead of from 0.04 to 0.1 per cent., as much as 0.48 per cent. of sugar)—which gives rise to *persistent excretion of sugar with the urine.* It is customary to distinguish two varieties of diabetes mellitus, and these are designated *idiopathic* and *symptomatic* respectively. In cases of *idiopathic diabetes mellitus* anatomic alterations in the body are wanting. The disorder is generally an *hereditary* one, which in some families is transmitted from generation to generation, or in other families occurs in alternation with central neuroses, particularly hysteria, neurasthenia, epilepsy, or mental disorders. Not rarely idiopathic diabetes mellitus results from *emotional disturbances*, as, for instance, speculations on the stock exchange, grief, anxiety of all kind, as well as excessive mental activity. Violent *mechanical agitation of the nervous system* in consequence of falls, blows, railway-accidents, and the like, are also capable of giving rise to diabetes mellitus.

Symptomatic diabetes mellitus develops in the sequence of some **nervous diseases.** Thus, it has been observed in association with hemorrhage, softening, suppuration, and neoplasms in the brain, involving the diabetic puncture-center on the floor of the fourth ventricle either directly or through a remote influence. At times diabetes mellitus occurs as a complication of diseases of the spinal cord, among which tabes dorsalis and multiple sclerosis may be mentioned. Diabetes mellitus occurs as a complication not only of anatomically demonstrable disease of the central nervous system, but occasionally also of **central neuroses,** as, for instance, neurasthenia, epilepsy, and mental disorders, and of exophthalmic goiter and akromegaly. Some cases of diabetes mellitus are dependent upon **chronic disorders of the stomach, intestine, liver, or pancreas.** It is noteworthy that long-continued **excessive indulgence in carbohydrates** may give rise to diabetes mellitus, whence this disease occurs frequently in countries (Italy) in which the inhabitants are especially fond of pastry and sweets. At times diabetes mellitus has been observed to develop in the sequence of **infectious diseases,** as, for instance, typhoid fever, influenza, cholera, syphilis, and malaria.

In a number of instances diabetes mellitus has been observed in *husband and wife*, in all probability because both have been exposed to the same injurious influences. Experience has shown that diabetes mellitus occurs but *rarely in children;* it generally develops *after the twentieth year of life. Men* are attacked more commonly than women, because they are more freely exposed to the injurious influences named. The *better classes of society* furnish more diabetic patients than the poorer classes. The statement that the disease has become more frequent in *recent times* is not incredible, because modern methods of living afford more favorable opportunities for nervous disturbances and injuries. Nevertheless, it should not be overlooked that at the present day patients are more carefully examined, and therefore disease is more frequently detected.

Symptoms.—The single trustworthy symptom of diabetes mellitus is the **presence of glucose in the urine**, although other morbid manifestations occur whose presence should always demand examination of the urine for glucose. Among such **suspicious symptoms of diabetes** may be mentioned excessive secretion of urine of a high specific gravity, increased thirst and appetite, with progressive emaciation, the formation of white spots (crystallized glucose) in places contaminated by urine, obstinate itching of the skin (cutaneous pruritus), chronic pruritus of the vagina, long-continued eczema, chronic furunculosis, the development of cataract before the fortieth year of life, an aromatic odor of the breath, frequently associated with states of coma, obstinate, and especially bilateral, neuralgia, abolition of the knee-jerks, and tabiform symptoms.

Of all the clinical manifestations the **changes in the urine** are of the greatest diagnostic importance. The *amount of urine* is almost always increased; daily amounts of between 5000 and 10,000 c.c. are not rare, but occasionally these may be considerably exceeded. Naturally, the patients are compelled to void urine more frequently than in health—so-called *pollakuria*—and especially sleep is much disturbed at night by the annoying vesical tenesmus. In view of the large amount passed, it is not surprising that the *color of the urine* is generally light yellow. It is noteworthy that bubbles of froth persist upon the surface of the urine for an unusually long time. This is a peculiarity that is also observed when the urine contains other foreign ingredients (albumin, biliary coloring-matter). The *specific gravity* is above the normal (from 1015 to 1020) in spite of the increased amount of urine, and it not rarely reaches and even exceeds the figures between 1030 and 1040. Nevertheless, exceptional cases occur in which the amount of urine is not increased and the specific gravity is diminished, and these have been designated *deceptive diabetes mellitus*. The *reaction of the urine* is acid, and increases in intensity on exposure to the air, because the glucose undergoes decomposition, and lactic acid results in consequence of its fermentation. The urine has a *sweetish taste*. Some patients make a practice of smelling their urine and thus estimating the degree of glycosuria. Before the urine is examined for sugar it should be examined for *albumin*, because this is capable of preventing the occurrence of the reactions for sugar. Should saccharine urine at the same time contain albumin, it should be boiled in a test-tube, several drops of dilute acetic acid be added for the precipitation of the albumin, and then be filtered. Tests for sugar may be readily made with the filtered urine thus freed from albumin.

Among the *tests for sugar* Moore's (Heller's), Trommer's, and Nylander's, and the fermentation-test suffice for the practitioner.

Moore's test for sugar consists in boiling urine to which has been added **one-third potassium or sodium hydroxid.** Urine free from sugar, thus tested, retains its original color unchanged or becomes somewhat more deeply yellow, while saccharine urine acquires a dark brownish-red, chestnut, or mahogany color. If nitric acid be added, drop by drop, the urine sputters and gives off an odor of burnt sugar (caramel or molasses).

In making *Trommer's test for sugar* a dilute solution of cupric sulphate (10 per cent.) and potassium hydroxid are required. From five to eight drops of the copper-solution mentioned are introduced into a test-tube filled one-quarter with urine, and then potassium hydroxid is added until the light-blue precipitate of cupric hydrate is wholly dissolved. Saccharine is distinguished from non-saccharine urine by the acquisition of a deep-blue color (blue-bottle, azure). If the urine be now heated, variations in the color take place in the heated portion, and the development of which is to be attributed to the fact that the sugar in alkaline solution takes up oxygen and reduces the copper-solution. If the sugar-test be performed slowly and carefully, a yellowish layer of cupric hydrate at first forms, which, on continued heating, acquires the reddish color of cupric oxid, and, finally, brown, lustrous metallic copper is precipitated in the test-tube. In order to guard against error in diagnosis the following considerations should be borne in mind: The urine should not be heated to the boiling-point, for, after long-continued boiling, reduction of the copper-salt takes place also in urine free from sugar. If only the upper layers of the urine are heated, the reduction and the discoloration that have been set in action extend also to the lower layers if the test-tube be removed from the flame and be permitted to stand at the temperature of the room. Decolorization alone is not distinctive, but also precipitation of granular masses must take place in the decolorized layers. Decolorization may be observed to take place also in urine free from sugar, if it contains uric acid and kreatin in abundance.

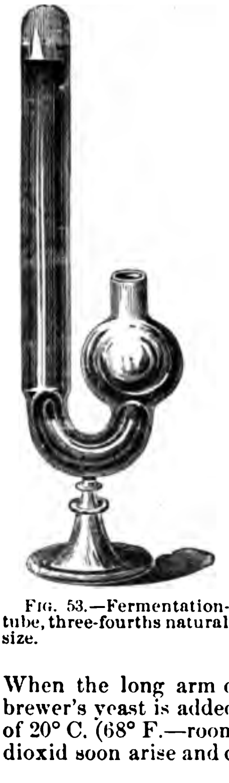


FIG. 53.—Fermentation-tube, three-fourths natural size.

Nylander's test for sugar requires the employment of a solution containing basic bismuth subnitrate, 2.0 (30 grains); potassium and sodium tartrate, 4.0 (60 grains); sodium hydroxid (8 per cent.), 100 (3½ fluidounces). If saccharine urine be boiled with this solution, it soon becomes from dark gray to black in consequence of reduction of the bismuth-salt. It should be borne in mind that urine free from sugar also undergoes the change in color if rhubarb, senna, antipyrin, salicylic acid, salol, camphor, or oil of turpentine has been ingested.

Of all the tests for sugar, the *fermentation-test* is the most reliable. It is to be recommended especially when the urine contains but small amounts of sugar, as, for instance, after the successful institution of an antidiabetic treatment, because, under such circumstances, the tests for sugar previously mentioned yield doubtful results. Glucose undergoes fermentation in the presence of yeast, and is decomposed into alcohol, glycerin, succinic acid, and carbon dioxid. In making the fermentation-test, tubes of the form indicated in Fig. 53 are employed. The tube is readily filled with urine by closing its orifice with the thumb and inverting it. When the long arm of the vessel is completely filled with urine a bit of brewer's yeast is added, and the tube is permitted to stand at a temperature of 20° C. (68° F.—room-temperature). In saccharine urine bubbles of carbon dioxid soon arise and collect beneath the dome of the long arm. The column of gas gradually becomes greater and greater and forces the urine downward, and even out through the opening in the tube. The generation of gas ceases after from twenty-four to thirty-six hours, because the sugar is wholly decomposed. In order to prove that the gas generated is carbon dioxid, potassium hydroxid is added to the urine, and the opening in the tube is closed with the thumb, while the tube is inverted. Under such circumstances the carbon dioxid is absorbed by the potassium hydroxid, and the thumb can be felt to be strongly sucked inward. In order to exclude all possibility of error in the performance of the fermentation-test this should be so carried out that three fermentation-tubes are employed, of which one is filled with urine, a second with a solution of glucose, and a third with water, a bit of yeast being added to each. It may, for instance, happen that the yeast is inactive or itself contains glucose. In the first event carbon dioxid will not be generated in any of the tubes, while active yeast will cause the development of carbon dioxid in the second tube. The freedom of the yeast from sugar will be demonstrated by the failure of carbon dioxid to develop in the third fermentation-tube.

In the clinical discrimination of diabetes mellitus it is important to know not only that the urine contains sugar, but also the *amount* eliminated. In the majority of cases the daily amount equals from 300 to 500 grams, although in some the amount may be much larger, as well as much smaller. The percentage varies generally between 5 and 10 per cent., although obviously it is not sufficient to determine the percentage alone, as the daily amount may be larger despite a low percentage than if the percentage be high. The amount of sugar must always be estimated from the total daily amount of urine, as great variations occur at different times of the day. In general the amounts of sugar in the urine are greater by day than by night. At the beginning of the disease, or with the advent of recovery, only certain portions of urine occasionally contain sugar, while others are wholly free from it. The larger the amount of carbohydrates ingested, the greater is the amount of sugar in the urine. Febrile infectious diseases occasionally cause disappearance of the sugar during their prevalence. The sugar also sometimes disappears from the urine in association with starvation.

For *quantitative determination of the amount of sugar*, titration with Fehling's solution is to be recommended on account of its accuracy, although it is somewhat inconvenient for the practitioner. The simplest method consists in the use of polarizing apparatus—so-called *saccharimeters*—although these instruments are quite expensive. A less expensive method of estimation consists in determining the specific gravity of the urine, then adding yeast, permitting fermentation of the sugar to take place for from twenty-four to thirty-six hours, and again observing the specific gravity. The difference in the specific gravity at the first and second observations is multiplied by the empiric figure 0.23, and the result represents approximately the percentage of sugar.

All of the tests and estimations for sugar have to do only with glucose, as this occurs most constantly and most abundantly in the urine in cases of diabetes mellitus. It is also known as urinary sugar, grape-sugar, and dextrose, and is one of the hexoses. Occasionally *fruit-sugar* (*levulose*) has additionally been found. This, although it is likewise a hexose and undergoes

fermentation with yeast, deflects the plane of polarization toward the left. Further, *pentoses* also are encountered with marked frequency in diabetic urine, together with hexoses. In isolated instances *muscle-sugar* (*inosite*), *glycogen*, *maltose*, and *dextrin* have been found.

Among the *remaining urinary constituents* especially the urea is increased in amount, so that instead of 30 grams, twice as much, and even four times that amount, may be present. Uric acid, hippuric acid, kreatin, chlorids, phosphates, and sulphates are also generally increased in amount. Not rarely the elimination of ammonia is greatly increased, and experience has shown that diabetic intoxication is likely to occur especially in such patients.

It would be incorrect to assume that the morbid alterations in the urine in cases of diabetes mellitus are confined to the elimination of sugar. Diabetic urine not at all rarely contains *diacetic acid, acetone, β-oxybutyric acid, formic acid,* and other *volatile fatty acids.* Urine that contains acetone generally possesses an aromatic odor suggestive of fruit, ether, or chloroform, while the *ferric-chlorid reaction* of Gerhardt depends upon the presence of diacetic, oxybutyric, and formic acids. Diabetic urine sometimes acquires a deep dark-red, cherry-red, or Burgundy-red color on addition of dilute ferric-chlorid solution of Rhine-wine yellow tint; but care should be taken to avoid confusion with the reaction of salicylic acid, the color resulting from which is a cloudy bluish-violet, and naturally occurs only in such cases of diabetes in which salicylic acid has been administered. The ferric-chlorid reaction of Gerhardt develops readily on the institution of too rigid an animal diet, and it is therefore always of serious significance, because such patients are exposed to danger of diabetic intoxication, in consequence of which death may result.

Diabetic patients complain generally of insatiable hunger and unquenchable thirst—**polyphagia and polydipsia**—conditions that are dependent in part upon disturbances of innervation, but in part, also, upon the metabolic processes going on. In spite of the large amounts of food ingested uncontrollable emaciation often occurs. Some patients, it is true, remain well nourished, so that a distinction has been made between lean and fat diabetics, and it has been assumed that in the former the disease is dependent upon lesions of the pancreas. The thirst is occasionally so urgent that children have been known to drink their urine at night. If diabetic patients and healthy individuals are given equal amounts of fluid to drink, the former eliminate the water with the urine more slowly than the latter. The large amounts of urine voided are responsible for the *dryness* and *stickiness of the mouth* of which diabetics generally complain, for the absence of a tendency to sweating, and for the presence generally of *dryness of the skin,* which usually feels rough and is covered with small epidermic scales (pityriasis tabescentium). The *general appearance* of a patient with diabetes depends upon the duration and the severity of the disease. Some present a fresh, almost blooming appearance, while others are collapsed, wrinkled, emaciated, and tired. The bodily temperature is frequently subnormal, and the **pulse** exhibits no peculiarity.

Complications occur with extreme frequency in the course of diabetes mellitus, and they may be either of inflammatory or of autotoxic origin. Intolerable and obstinate *itching of the skin—cutaneous pruritus*—occurs not rarely. In other instances the patient suffers from *chronic eczema* and *furunculosis*. There is a marked tendency to *gangrene of the skin*, which may result from slight wounds, or even without appreciable cause. *Disorders of the eye* often occur. Most frequently *opacity of the crystalline lens—cataract*—develops. Occasionally neuroretinitis, resembling that of nephritis develops, but a number of other inflammatory changes in the eye have been observed. It is noteworthy that in diabetic patients the teeth are rapidly lost in consequence of caries. This manifestation is attributed to the fact that the sugar is decomposed in the secretions of the mouth, and the resulting lactic acid dissolves the enamel of the teeth, and thereby renders them more accessible to the invasion of lower forms of life. The saliva, therefore, frequently yields an acid reaction, and readily gives rise to inflammation of the buccal mucous membrane—*stomatitis*. Sugar has been frequently, though not constantly, demonstrated in the secretion of the parotid gland.

The *lungs* are attacked with exceeding frequency. Many diabetic patients acquire *pulmonary tuberculosis*, to which they succumb, or *pulmonary gangrene*. It is noteworthy that with the latter the sputum is only slightly if at all offensive. Disease of the *heart* and the *blood-vessels* occurs but seldom, although it is maintained that diabetes mellitus creates a tendency to arteriosclerotic changes. At times attacks of angina pectoris or cardiac asthma occur.

Dilatation of the stomach is not rarely encountered in diabetic patients, in consequence of excessive ingestion of food. The functional activity of the stomach may, under such circumstances, be wholly unimpaired. Generally there is a tendency to *constipation*, and the stools are frequently hard and dry. Profound disease of the pancreas is revealed in some cases by fatty diarrhea—*steatorrhea*. Occasionally the *liver* is enlarged and hard, in consequence partly of decomposition of fat, and partly of connective-tissue hyperplasia. *Albuminuria* is by no means rarely encountered in cases of diabetes; and occasionally signs of *acute* or *chronic nephritis* appear. *Impotence* gradually develops in cases of diabetes, inasmuch as the generation of spermatozoids ceases. At the beginning of the disease, it is true, I have often observed increased sexual desire. Women are often harassed by intolerable and obstinate *itching of the vagina—pruritus of the vagina*—which is dependent upon the presence of molds upon the mucous membrane of the vagina. Eczema and furuncles of the labia, and even gangrene, may readily occur. Further, inflammation of the prepuce also readily develops in men, and masses of mold (leptothrix) may collect beneath the part.

Among *nervous disturbances* obstinate *neuralgia* should first be mentioned. *Sciatica* occurs with especial frequency, and its diabetic origin should particularly be suspected if it be bilateral in distribution. Occasionally signs of *polyneuritis* appear. This is attended with abolition of the knee-jerks and with peripheral motor paralysis and cutaneous anesthesia. The symptoms of *peripheral pseudotabes* develop, as manifested by ataxic gait, tardy pupillary reaction, and swaying when the eyes are closed. Some patients complain of attacks of *cramp in the calves.* Arteriosclerotic changes in the cerebral arteries occasionally lead to *cerebral hemorrhage.* At times *mental disease* is superadded to diabetes.

One of the most serious dangers for the diabetic patient consists in *diabetic intoxication,* generally designated also *diabetic coma.* This is considered the result of *auto-intoxication* with abnormal metabolic products. By some β-oxybutyric acid especially is considered as the toxic agent, and diabetic auto-intoxication is accordingly thought to be an acid intoxication. One can frequently anticipate the advent of diabetic intoxication from the fact that the urine yields a marked ferric-chlorid reaction of Gerhardt, and that the urine and the expired air of the patient occasionally give off so pronounced an aromatic odor of apples or ether that the disease can be recognized by the sense of smell on entering the sick-room. The patients are more or less suddenly seized with coma, frequently breathe loudly and noisily, and exhibit especially unusually deep respiratory movements. Occasionally they attempt to maintain themselves upon their feet, but they stagger about like a drunken person. In some violent delirium occurs, and even maniacal states. Death occurs at times within a few hours amid signs of cerebral or cardiac paralysis, but in other instances only after the lapse of a few days. At times transitory recovery ensues, possibly to be followed in a short time by relapse and a fatal termination.

Diabetes mellitus is generally a *chronic disease,* whose duration not rarely extends over many years if a rigid diet be observed. Cases that terminate fatally within a few weeks are rare. The principal dangers that threaten the diabetic patient consist in pulmonary tuberculosis and pulmonary gangrene, in phlegmons and gangrene of the skin, and in diabetic intoxication. With regard to the *relations of diabetes to other diseases* it is especially to be mentioned that the obese, the gouty, those suffering from renal and biliary calculi, are frequently attacked by diabetes mellitus.

Diagnosis.—The recognition of diabetes mellitus is easy if one is competent to make the tests for sugar. In the presence of sugar in the urine the possibility of transitory *glycosuria* or *mellituria* is at most to be considered, although this persists for only a few hours, and at longest a few days. At the beginning of diabetes mellitus sugar occasionally appears only at times and in certain portions of the urine—*intermittent diabetes mellitus*. Under doubtful circumstances the patient should be permitted to take a mid-day meal containing considerable carbohydrates, particularly sugared fruits, sweet farinaceous food and pastry, and the urine passed from four to six hours later examined for sugar. Should the urine then also be found free from sugar the exclusion of diabetes mellitus would be justifiable. It is extremely important to know whether the patient is suffering from *diabetes mellitus of slight, or moderate, or profound degree*, and this can be determined from the influence of a strictly animal diet upon the elimination of the sugar. In the cases of slight degree the sugar disappears from the urine in from one to three days of an animal diet; while in a case of moderate severity fourteen days will be required; and in a case of profound degree, finally, although the amount diminishes, the sugar does not wholly disappear from the urine. Further, these three gradations cannot always be sharply discriminated from one another, and it may happen that in a case of slight degree transitory periods occur in which symptoms of moderate severity, or of profound degree, appear. The *etiologic diagnosis* further is important, for when syphilis has been the cause cure has been reported from mercurial treatment. If pseudotabic symptoms predominate care must be taken to avoid confusion with tabes dorsalis. The demonstration of sugar in the urine generally affords a speedy means of differentiation, although diabetes mellitus occasionally occurs as a complication of tabes dorsalis.

Anatomic Alterations.—Anatomic alterations distinctive of diabetes mellitus are not known, so that in cases of idiopathic diabetes mellitus no striking peculiarity is found in the body. In cases of symptomatic diabetes mellitus lesions will be found at times in one, and at other times in another organ, in accordance with the causative factors. Attention has for a long time been directed especially to atrophy and other alterations in the pancreas as not rare conditions. On *microscopic examination of the viscera* alterations in the kidneys are not rarely found. The epithelial cells of the convoluted uriniferous tubules are occasionally involved in fatty degeneration, or they are in a state of coagulation-necrosis, and the epithelial cells of the loops of Henle are converted into swollen, transparent vesicles, involved in hyaline degeneration. The presence in them of glycogen can be demonstrated by means of iodin. Deposits of glycogen have also been described in the myocardium. At times the liver-cells also are characterized by the presence of considerable amounts of glycogen.

Opinions are sharply divided as to the *pathogenesis of diabetes mellitus.* Obviously, elimination of sugar with the urine may take place in consequence of excessive ingestion of carbohydrates, because the liver is incapable of converting all of the sugar into glycogen, and retaining it. Such glycosuria can be experimentally developed in healthy individuals by the administration of more than 200 or 300 grams of grape-sugar—so-called *alimentary glycosuria.* Saccharine urine will be excreted also if the liver has wholly lost its power of converting even small amounts of ingested sugar into glycogen, for sugar, which, without previous transformation into glycogen, gains entrance directly into the blood, is largely again eliminated with the urine, unused. There results in this way a *hepatogenous glycosuria.* It is naturally conceivable also that in spite of normal formation of sugar those ferments may be absent from the blood and the tissues that are necessary for the decomposition and utilization of the sugar in the work of the body. The blood in consequence remains unusually rich in sugar, and the resulting hyperglycemia gives rise to diabetes mellitus. Such a condition is one of *hematogenous glycosuria.* In the development of this variety of glycosuria the pancreas especially is believed to be involved. After *complete* removal of the pancreas diabetes mellitus has been observed to develop in animals, and this is explained by some physicians on the assumption that one of the functions of the pancreas is to form a glycolytic ferment. Reference to a *neurogenous variety of glycosuria* should not be omitted. This develops temporarily in animals, among other conditions, after injury to a definite portion of the fourth ventricle, so-called puncture. Possibly it is dependent upon vasomotor disturbances in the liver, induced from the portion of the brain mentioned through the intermediation of the sympathetic nerve. In all probability there are still other causes for glycosuria. Thus the glycosuria that can be developed artificially by the administration of *phloridzin* has been referred to renal causes—*nephrogenous glycosuria,* and some clinicians believe in the possibility also of *myogenous glycosuria.* Especial difficulties will, however, arise in attempting to refer the individual case of diabetes mellitus to its etiologic factors.

Prognosis.—Diabetes mellitus is a *serious disease,* which is but seldom susceptible of permanent cure, and even then will require careful regulation of the diet. The prognosis varies in accordance with the **age.** Diabetes mellitus is prone to pursue a rapidly fatal course, particularly in children. As may be understood, the prognosis will be governed by the **degree of the diabetes mellitus,** and, as a matter of course, the prognosis will be better in cases of the milder grade than in those of moderate severity and of the severe grade. The ferric-chlorid reaction of Gerhardt is of unfavorable prognostic significance, because it frequently precedes diabetic auto-intoxication. Among the *complications* the prognosis is rendered unfavorable particularly by pulmonary tuberculosis, pulmonary gangrene, and gangrene of the extremities.

Treatment.—The indication for **causal treatment** is but seldom present. If the disease has been preceded by syphilis, a course of treatment with mercurial inunctions and the internal administration of potassium iodid will be prescribed, and recovery has occasionally been reported from such measures. In the **symptomatic treatment** subordinate and doubtful significance is to be attached to drugs. In any event the greatest importance is to be attached to the **diet.** The guiding principle in an antidiabetic diet consists in withholding from the patient carbohydrates as largely as possible, but, on the other hand, permitting sufficient animal food and fats to supply the needs of the body in calories. In entering upon an animal diet the transition should not be too rapid and abrupt, as otherwise diabetic intoxication may readily result. Instead of sugar the patient should use **saccharin** (sulphinid of benzoic acid). The patients tolerate least well the withdrawal of bread, inasmuch as no palatable bread free from starch is as yet known. Generally 100 or even 150 grams of Graham bread may be permitted daily, as a diabetic patient with some sugar in the urine and progressive increase in weight is far better off than one in whom the urine has been freed from sugar, but whose bodily weight is gradually diminishing, and who is threatened by the dangers of diabetic auto-intoxication. An alkaline water, such as that of Selters, Bilin, Giesshübel, Ems, or Vichy, should be used by the diabetic as a **beverage.** To maintain the skin in good condition two **baths** (35° C.—25° R.—95° F.) weekly may be recommended. Attention should be directed to the **care of the mouth,** which should be thoroughly rinsed (solution of aluminum acetate, 1.0 : 200—as a mouth-wash) after each meal. The diabetic patient will do well to protect himself from the influence of cold by wearing woollen **underclothing. Wounds and operative measures** are to be carefully avoided in consequence of the danger of gangrene, and in any event they should be treated with the most rigid antiseptic precautions. Of late, special *sanatoria* for the treatment of diabetics have been erected, which have the advantage, among other things, of teaching the patient how to live.

A brief consideration of the *permitted and the forbidden articles of food* may yet be given in this place.

There may be *permitted:* meat of all kinds (fresh meat, smoked meat in the form of ham, sausage, tongue, corned meat, pickled meat, fowl, game, fish, oysters, lobster, and shell-fish), eggs, cheese, unsweetened fruit-juice and gelatin; butter, bacon, cream, cod-liver oil, and olive-oil; green vegetables, such as cauliflower, red cabbage, spinach, green asparagus-tips, green beans, turnip-tops, lettuce, white cabbage, endives, water-cress, radishes, mushrooms; almonds, nuts; alkaline waters, white wine, red wine, unsweetened lemonade from lemons or lactic acid, unsweetened tea and coffee.

There are *forbidden:* honey, ordinary bread, cake, farinaceous food, sago, arrowroot, noodles, maccaroni, oatmeal, barley-grits, whey, chocolate, beer, sweet wine, champagne, alcohol, liqueurs, compot, red beets, onions, radishes, celery, rhubarb, green pease, cucumbers, and chestnuts.

Drugs, without rigid restriction of the diet, are without utility. In some cases the amount of sugar in the urine diminishes after the administration of *narcotics,* as, for instance, opium, 0.02 (⅓ grain) thrice daily. Also *nervines* (arsenic, bromids, antipyrin) have been employed and recommended. *Disinfectants* (carbolic acid, salicylic acid), if they have any influence whatever in diminishing the amount of sugar in the urine, do so by causing impairment of appetite and restriction in the amount of food taken. **Ferments** (brewers' yeast, diastase, lab) also have been employed. Recently **organotherapy** has been resorted to, and pancreatic extract has been administered. Courses of treatment at the **springs** have been much prescribed, particularly at Carlsbad, Neunahr, and Vichy. I have observed the sugar to disappear from the urine in many cases of diabetes after a course of treatment at Carlsbad, but probably rather in consequence of regulation of the diet than from the effects of the alkaline waters. It is true that generally after a time the sugar reappeared, because the patients lived less carefully at home, and in this way the course of treatment, in some of the cases under my observation, was repeated almost annually with good results.

Complications should be treated in the usual manner. For the relief of diabetic intoxication, intravenous infusion of sodium carbonate (from 3 to 5 per cent.), sodium bicarbonate (5 per cent.), and sodium chlorid (0.6 per cent.) have been recommended, although the results have not as yet been permanent. Occasionally improvement has been observed after the administration of a vigorous laxative.

DIABETES INSIPIDUS (POLYURIA).

Etiology.—Diabetes insipidus is characterized by two distinctive symptoms—an *increased amount of urine of diminished specific gravity and increased thirst.* In the same way as with diabetes mellitus, two varieties of diabetes insipidus, *idiopathic* and *symptomatic,* may be distinguished. *Idiopathic diabetes insipidus* occurs in some families as an *hereditary disorder.* Under such circumstances it has occasionally been observed in alternation with other central neuroses (hysteria, epilepsy, neurasthenia, psychopathy), or, occasionally, also with diabetes mellitus. At times idiopathic diabetes insipidus develops in the sequence of **emotional disturbances. Concussion of the body,** also, in which the central nervous system participates, may be a cause of the disease. In some cases it has been observed to develop in the sequence of **infectious disease,** as, for instance, typhoid fever, influenza, and syphilis. The designation *toxic diabetes insipidus* has been applied to such cases as occasionally develop after the use of digitalis or other diuretics. Alcoholism and saturnism also may be causes of the disease. *Symptomatic diabetes insipidus* is generally the result of anatomically demonstrable **disease of the nervous system.** From experiments upon animals it is known that upon the floor of the fourth ventricle, near the point puncture at which is followed by glycosuria, there is another point injury of which is followed by increased elimination of urine, and in conformity with this fact simple polyuria has been observed in association with softening and new-growths in the region named. Diabetes insipidus but rarely occurs in children, and is less common than diabetes mellitus. *Men* are attacked much more frequently than women.

Symptoms.—In some patients **premonitory symptoms—prodromes**—appear, consisting especially in headache, vertigo, malaise, irritability, disturbed sleep, alterations in appetite, and the like. In most instances the attention of the patient is attracted to his disease by the increased **amount of urine,** which necessitates increased frequency of micturition—*pollakiuria*—and greatly disturbs rest at night. The *amount of urine* voided in twenty-four hours may reach from 5000 to 10,000 c.c., and even more, and the fluid is generally of a pale-yellow, almost watery *color*, and is characterized by a low *specific gravity*, which in a case under my care was as low as 1000.5. The urine is feebly acid in *reaction.* The percentage of normal urinary constituents is diminished, but the daily amount eliminated is not reduced, but, on the contrary, is often increased. In some cases *inosite* has been found in the urine, and this can be abstracted experimentally from the body by the ingestion of large amounts of water and increased elimination of urine. The patients are harassed by great thirst—**polydipsia**—and therefore generally complain of *dryness and stickiness of the mouth.* In consequence of the large loss of water through the kidneys, the skin exhibits but slight tendency to *sweating*, and it is generally dry, shrivelled, and scaly. The muscular tissues also are shrivelled and dry. Generally **pallor of the skin** and **emaciation** develop gradually. The patients attract attention not rarely in consequence of their irritable, **nervous manner,** and they complain frequently of headache, vertigo, and neuralgia. Occasionally the knee-jerks are enfeebled, or even abolished. Neuroretinitis and paralysis of the ocular muscles also develop occasionally, but, in contradistinction from diabetes mellitus, cataract does not. The **bodily temperature** not rarely is remarkably low, and the patients complain of chilliness. The disease pursues a *chronic course*, and at times persists for many years. *Death* generally results in consequence of excessive exhaustion.

Attention has often been called to a relation between diabetes insipidus and diabetes mellitus, as exhibited in the fact that both diseases may be transmitted by heredity in some families, so that some of the members suffer from diabetes mellitus and others from diabetes insipidus. It may also happen that diabetes mellitus is preceded by diabetes insipidus, so that a subsiding diabetes mellitus is followed by diabetes insipidus. In isolated instances periods attended with diabetes mellitus alternate in the same patient with periods attended with diabetes insipidus. In some cases of diabetes insipidus, finally, the patients exhibit an undue predisposition to alimentary glycosuria.

Diagnosis.—Diabetes insipidus is easy of recognition. Although **diabetes mellitus** is attended with polydipsia and polyuria, the specific gravity of the urine is increased and the secretion contains sugar. In the presence of **chronic contracted kidney** also the urine is abundant and of low specific gravity, but it contains albumin and tube-casts; and, besides hypertrophy of the heart, a wiry pulse and albuminuric retinitis are also present. Increased elimination of urine of diminished specific gravity occurs also in connection with **diseases of the urinary passages** through reflex influences, but under such circumstances the symptom is a secondary phenomenon, which disappears after the relief of the primary disorder. Increased secretion of urine occurs not rarely as a transitory manifestation in the sequence of infectious diseases—the *polyuria of convalescence*—but this disappears within a few days. **Nervous disorders** (emotional disturbances and overexertion, hysteria, neurasthenia, cohabitation, cerebral hemorrhage) not rarely give rise to transient and insignificant polyuria. An attempt has been made to enforce in the clinical picture of diabetes insipidus a distinction between *primary polydipsia* with secondary polyuria, and *primary polyuria* with secondary polydipsia. A study of the history will often leave one in doubt. It has been recommended that the patient be deprived of water for a time. In a case of primary polydipsia the amount of urine will thus be greatly diminished, while in a case of primary polyuria, although the amount of urine be diminished, it nevertheless remains large.

Anatomic Alterations.—No anatomic alterations distinctive of diabetes insipidus are known, so that such lesions may be wholly wanting in a case of idiopathic diabetes insipidus. Nothing, likewise, is therefore known with regard to the *nature of the disease*, although there is much evidence in favor of a functional disorder of the secretory nerves of the kidneys.

Prognosis.—Diabetes insipidus proves, as a rule, to be an incurable disease, and to this degree the prognosis is not favorable. Nevertheless, the affection frequently persists for many years.

Treatment.—**Causal treatment** will probably be indicated only if *syphilis* is the cause of the disorder, and it will then consist in inunctions with mercurial ointment (5.0—75 grains—thrice daily) and the internal administration of potassium iodid (5.0 : 200 —75 grains: 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily). Among **symptomatic remedies**, *nervines* (bromids, valerian), *narcotics* (opium), and *astringents* (lead acetate, extract of ergot) have been recommended. The *galvanic current* and *preparations of iron* also have been employed. In my experience antipyrin has been most effective, and in some cases also phenacetin.

℞ Antipyrin,	1.0 (15 grains);
Sugar	0.5 (7½ ").—M.

Make 10 such powders.
Dose: 1 powder every three hours.

For the relief of the distressing thirst the coöperation of the patient is necessary. In addition, the pharyngeal mucous membrane should be rinsed several times daily with cool water, and small bits of ice should be permitted to melt slowly in the mouth.

Among the *less common disorders of metabolism* are the following:

Cystinuria, which occurs most frequently in men, and has occasionally been observed after *infectious diseases* (articular rheumatism, syphilis). The urine frequently contains six-sided plates of *cystin-crystals*, which, in contradistinction from similar crystals of uric acid, do not respond to the murexid-test. If all of the cystin is dissolved in the urine, acetic acid, in which the cystin is insoluble, should be added, when crystals of cystin will be precipitated. Cystin is a sulphurous body, and therefore urine containing it frequently gives off on decomposition the *odor of hydrogen sulphid*. The latter should especially arouse suspicion of cystinuria if the urine is free from albumin. As urine containing cystin is rich in *diamins*, cystinuria has been considered an *infectious disease*, in the course of which bacteria cause the development of abnormal metabolic processes in the intestine. The principal danger of cystinuria consists in its tendency to the formation of *cystin-calculi*. It is well to disinfect the intestine with *salol* (1.0—15 grains—every two hours), *calomel* (0.5—7½ grains) or repeated *enteroclysis*, and to administer internally *nitric acid* or *nitrohydrochloric acid*. In addition, the employment of fresh *meat* and *green vegetables* is to be recommended.

Alkaptonuria is revealed by the development of from a brownish to a Burgundy-red color in the urine on standing in the air, and which appears at once when potassium hydroxid is added. If the urine be agitated in a test-tube with potassium hydroxid, and the opening of the tube be closed with the thumb, the thumb will be sucked inward in consequence of absorption of oxygen. The urine will reduce an alkaline solution of copper and an ammoniacal solution of silver in the cold; but, in contradistinction from glucose, it has no influence upon the plane of polarization. Several substances (pyrocatechin, glycosuric acid, uroleucinic acid, homogentisic acid) are known to be capable of inducing the clinical picture of alkaptonuria. Further, the urine also stains the linen brown and renders it soft and friable. Morbid manifestations do not appear. The condition has been observed after the use of salicylic acid and in cases of pulmonary tuberculosis. Occasionally several members of the same family have suffered from alkaptonuria.

Oxaluria is considered by some physicians to be a metabolic disorder. It is characterized by the presence of calcium oxalate in large amount in the urine (octahedral, envelop-shaped crystals) and by dyspeptic and nervous symptoms. Quantitative determinations of the oxalic acid eliminated in the urine are as yet wanting, and the condition is of doubtful significance.

The last statement is applicable also to *phosphaturia*, which occurs especially in neurotic individuals, and is attended with turbidity of the urine, which increases on application of heat, but, in contradistinction from albuminuria, is wholly dissipated on addition of acetic or nitric acid. For the relief of phosphaturia hydrochloric acid (5.0 : 200—75 minims : 6½ fluidounces; 15 c.c.—1 tablespoonful—every two hours) should be used for a long time.

RACHITIS.

Etiology.—Rachitis is an extremely frequent *disease of childhood*, in which the bones have lost the faculty of taking up and retaining calcium-salts from the food, so that the *bones exhibit* *abnormal pliancy.* Hand in hand with these alterations *inflammatory processes* take place *at the points of growth of the bones,* the most marked result of which is *enlargement of the epiphyses.* It was for a long time believed that the disease is a result of **improper feeding and mode of life,** because such children were attacked with especial frequency as were not brought up at the mother's or at a wet-nurse's breast, and also not upon animal milk, but had received substitutes for milk and flour-paps as food. The disorder is therefore encountered with especial frequency among the poorer classes in the community, in whom insufficiently lighted, damp, and overcrowded rooms and a **deficiency of activity in the open air** operate as further injurious influences. It has recently been maintained that rachitis is an *infectious disease,* although bacteria or parasites capable of causing the disease have not as yet been discovered. Rachitis has often been considered an *hereditary disease,* but the injurious influences responsible for its occurrence are so widespread that its development in several children in the same family, and even in the parents, is not with certainty indicative of hereditary transmission. The possibility of inheritance would be more probable if it should be found that children frequently present rachitic alterations in the bones already at birth. Among the *contributory causes* for the development of rachitis may be mentioned *marriage of the parents in advanced years, syphilis* and *chronic debilitating diseases in the parents, rapidly successive deliveries,* and the *bearing of a large number of children.*

Symptoms.—The symptoms of rachitis make their appearance most frequently *between the sixth month and the close of the second year of life.* Cases of *fetal* and *congenital rachitis* have also been reported, although it is still doubtful whether the alterations in such cases were really rachitic. The disorder *develops* slowly and insidiously. Frequently the rachitic changes in the bones have been preceded by *chronic diarrhea.* The children under such circumstances emaciate progressively, the skin becomes pale and deficient in fat, the cutaneous veins become dilated and tortuous, and the abdomen is frequently distended. Occasionally the first symptoms of rachitis consist in **disorders of dentition.** Children in whom no tooth has appeared at the end of the first year of life are generally rachitic. **Marked sweating of the head** also is considered a symptom of rachitis.

The **rachitic alterations in the bones** are generally present in most of the bones, although not rarely more marked in some than in others. Most frequently they appear earliest in the skull, whence they extend to the trunk and the extremities. *Rachitis of the bones of the head* causes first a change in the *shape of the skull.* The cranial cavity, in comparison with the facial portion of the skull, appears unusually capacious. At the same time the frontal bone rises almost vertically, while the occiput is expanded and flattened. On transverse section the skull presents a quadrangular rather than a circular shape. The *fontanels*, of which the posterior and smaller should be closed in healthy children at the end of the sixth month in life, and the anterior and larger in the middle of the second year, at times remain open up to the twenty-fourth month and later. At the same time they are conspicuous for their unusual size, so that, for instance, the anterior fontanel occasionally extends from the sagittal suture to the middle of the frontal bone. The borders of the cranial bones bounding the fontanels are thickened and everted. Should ossification of the fontanels subsequently take place, this is often effected through the development of *supernumerary* (*Wormian*) *bones*. The *frequent thinning of the cranial bones* is noteworthy. This may attain so marked a degree, especially in the occipital region, that the skull in this situation can occasionally be indented like a sheet of cardboard or parchment, with a crackling sound. Great care, further, must be taken in making such observations, as in the application of too great force unconsciousness and general clonic convulsions may be induced through pressure on the brain. At times the attenuation is so great that bony tissue is wanting, and deficiencies in the skull have resulted at some points upon the occiput. In the presence of the changes described the designation *soft occiput* or *craniotabes* also is employed, and the development of the condition is attributed to the pressure of the brain upon the soft bony tissue in the dorsal decubitus. The occiput is often bald, in consequence of persistent pressure in the dorsal position and of profuse sweating of the head. On auscultation of the skull a vascular murmur —so-called **cerebral blowing**—synchronous with the carotid pulse, is not rarely heard, originating in the carotid canal of the petrous bone, because this is disproportionately small as compared with the size of the artery.

Rachitic alterations occur also in the facial portion of the skull. The *eruption of the teeth* especially is interfered with. Not only, as already mentioned, does this take place late, but the teeth appear in irregular order, readily become carious, or irrupt in unusual situations, as, for instance, through the anterior alveolar wall. They are also frequently excessive or deficient in number, and exhibit a terrace-like arrangement upon their broad surface, where the enamel is wanting. The *inferior maxilla* loses its arched shape, and acquires a somewhat six-sided form, while the *superior maxilla* appears depressed in the situation of the malar bones, and is increased in length. In the lower jaw the alveolar processes frequently acquire an abnormal position, their free border being directed backward, so that the teeth in consequence also assume an improper position.

The chest generally exhibits *enlargements at the costochondral junction* which not rarely appear as bulbous prominences beneath the skin, and which, even when not visible, are palpable to the fingers. This condition has been designated the *rachitic rosary*, because it forms a chain passing from above and inward, downward and outward. The ribs frequently exhibit laterally deep depressions inward, resulting in part from the pressure of the nurse's hands in lifting the child. At the same time the sternum often projects acutely forward, so that the designation *rachitic chicken-breast* (*pectus gallinaceum*) or *rachitic keel-breast* (*pectus carinatum*) has been employed, the transverse section of the thorax being comparable with the form of a bird's breast or of the keel of a ship. The *sternum* itself, also, not rarely exhibits deformity. Sometimes it projects acutely forward at the junction between the body and the ensiform cartilage; at other times it forms a deeply depressed gutter. Deflections to one side also are not at all uncommon occurrences. The *clavicles* frequently exhibit marked enlargements at either extremity, and similar changes are found also at the borders and angles of the *scapulæ*. The *vertebral column* is often the seat of curvature in consequence of rachitis. Posterior curvature—*kyphosis*—is common, and in contradistinction from that due to tuberculosis of the vertebræ, the prominence resulting from rachitis does not form an acute angle, but is generally arched. Lateral curvature of the vertebral column (scoliosis) and anterior curvature (lordosis) also occur, and frequently also a combination of several varieties of curvature.

Rachitis of the bones of the pelvis often gives rise to a *flat contracted rachitic pelvis*, the symphysis pubis and the promontory of the sacrum being closely approximated, so that the conjugate diameter is diminished. Another variety of rachitic pelvis is *heart-shaped*, the symphysis pubis projecting sharply forward like a proboscis, while the acetabular region on either side is pushed greatly inward. Rachitic changes in the pelvis are serious in the case of girls, as subsequently they not rarely cause obstruction in labor.

In the *extremities* the *enlargement of the epiphyses* is especially conspicuous. The inferior epiphyses of the radius and ulna particularly are often so greatly increased in size that a deep depression results between them and the carpal bones, whence the disease has been designated also double joint. Corresponding alterations take place frequently between the tibia, the fibula, and the tarsal bones. In addition, there may be *curvature of the bones*, which is generally more pronounced in the legs than in the arms, because the lower extremities must support the weight of the body. The curvature under such circumstances becomes especially marked, if, notwithstanding their disease, the children are urged to remain upon their feet often and for long periods of time. Many children, it is true, have a natural inclination to walk and to stand as little as possible, and often incipient rachitis is indicated by the fact that the children begin to walk only late, or that, after having learned to walk, they cease to do so. Most frequently the legs become saber-shaped or O-shaped—genua vara—the bones acquiring a convex curvature outward; but in other children the convexity of the curvature is directed inward —genua valga—or forward or backward. Such curvatures interfere with walking, and render the gait waddling and clumsy, and in more pronounced degree as the articular ligaments generally are greatly relaxed. At times the deformities are so marked that infraction results. Pain is generally wanting; only rarely is pain in the bones present on pressure or also spontaneously.

In correspondence with the pallid appearance of rachitic children, **leukocytosis** can generally be demonstrated in the blood; nucleated red corpuscles also have occasionally been found. Frequently **enlargement of the spleen** occurs, and this occasionally attains a marked degree. The **urine** exhibits no constant change, and especially the earlier statements with regard to the presence of increased amounts of calcium and phosphoric acid, and the occurrence of lactic acid, have not been confirmed. On the other hand, the **feces** contain an unusually large amount of calcium-salts.

Rachitis generally pursues a *chronic course*. Cases of *acute rachitis* with a febrile course are rare. Rachitis often occurs in association with *scrofulosis*. *Hydrocephalus* and *spasm of the glottis*, and also scorbutus, so-called Barlow's disease, have been observed as complications.

Prognosis.—The prognosis of rachitis is not unfavorable, as the disease is itself scarcely attended with immediate danger to life, and the curvature of the bones slowly undergoes gradual involution to a certain degree. The prognosis is more serious in the case of girls, as rachitis of the pelvis may later in life become a source of danger in the act of parturition. An unfavorable outcome of the disorder depends rather upon the *complications*. Rachitic children are markedly predisposed to inflammation of the bronchial mucous membrane and the lungs, to miliary tuberculosis and caseous tuberculosis, and to chronic and exhausting diarrhea.

Diagnosis.—The recognition of rachitis is easy. The disorder is distinguished from **osteomalacia**, which also gives rise to abnormal pliancy and curvature of the bones, by the fact that the former occurs in children and the latter in *adults*, and that in rachitis the bones *remain soft*, while in osteomalacia the bones previously hard *become soft*.

Anatomic Alterations.—The **softness of rachitic bones** can frequently be recognized with especial distinctness in the skull, which can be cut with readiness. In the long bones, as a matter of course, the **epiphyseal enlargement** is especially conspicuous, being dependent upon changes in the *epiphyseal cartilage.* In the portion of the epiphyseal cartilage immediately adjacent to the epiphysis—the so-called *zone of hyperplasia of the cartilage-cells*—such marked proliferation of cartilage-cells has taken place that these are collected close together in large masses and groups, and in consequence of want of room have displaced the epiphyseal cartilage to one side. In the *developmental and hyperplastic zone* beyond the proliferating zone toward the diaphysis, in which in healthy epiphyseal cartilage the cartilage-cells are arranged in regular lines, and in the oldest layers are enlarged, it will be observed that such an orderly arrangement of the cartilage-cells is partly wanting, that the interstitial tissue has acquired a fibrous character, and that, besides, contrary to the rule, medullary spaces containing blood-vessels are present, which have penetrated from the third layer of the epiphyseal cartilage yet to be mentioned, the zone of preliminary calcareous infiltration of the developmental zone, and open here and there in groups of cartilage-cells. The presence of a number of calcified areas in the developmental zone must also be considered as abnormal. In the zone of *preliminary calcareous infiltration* previously mentioned, the third section of the epiphyseal cartilage immediately adjacent to the diaphysis, the unusually extensive formation of medullary spaces is conspicuous, so that the tissue is porous and extremely vascular. Calcification of the interstitial tissue occurs irregularly and imperfectly. While the longitudinal growth of the bone takes place from the epiphyseal cartilage, the increase in thickness depends upon **developmental processes in the periosteum.** These also are disturbed in cases of rachitis, abnormal proliferation, marked development of vascular spaces, and deficient calcification occurring, with the development of porous, osteoid tissue. The thickness of the bone becomes diminished because osseous tissue is but imperfectly formed from the periosteum, while it is absorbed in the usual manner through the medullary cavity. It has, probably not incorrectly, been emphasized of late that inflammatory processes are operative in the rachitic changes in the bones described.

Rachitic bones are extremely *light in weight,* and their specific gravity also is diminished. They contain small amounts of calcium and of inorganic substances generally, while they are rich in organic substances. Nothing of a definite nature is at present known with regard to the *pathogenesis of rachitis.* Nevertheless, the imperfect calcification of the bones is not the result of a *deficiency of calcium in the food,* because it has been demonstrated that the food of an infant contains a sufficient amount of calcium-salts. The view has gained many adherents according to which in cases of rachitis abnormal *acids are present in the blood,* which retain the calcium-salts in solution, and prevent their deposition in the bones. Lactic acid especially has been thought of, and its formation attributed to the decomposition of the milk in the intestine resulting in connection with diarrhea frequently observed in rachitic children. Attention has recently been directed to the abundant presence of calcium in the feces, and an attempt

has been made to attribute rachitis to *deficient absorption of calcium-salts from the intestine.* If, however, it is correct that rachitis, or, at least, the predisposition to rachitic changes in the bones, is already congenital, it is likely that the basis of the disorder is to be sought for in a *morbid tendency on the part of the bones,* whose nature, it is true, is as yet wholly unknown. The adherents of the view that rachitis is of parasitic origin would have to assume that the bacterial poisons or toxins resulting from the action of the causative bacteria would be responsible for this questionable tendency on the part of the bones.

Treatment.—Preventive measures are of great importance in the *prophylaxis* of rachitis. They include an *appropriate diet* and a *suitable mode of life for the children.* Infants should be nursed at the mother's or at a wet-nurse's breast, or, at any rate, should receive animal milk, while substitutes for milk should be rejected for purposes of permanent nourishment. Of late, reports have accumulated showing that nourishment with sterilized animal milk likewise is frequently followed by rachitis, and in any event lime-water should be added to such milk. It is important to send the children into the open air as often as the weather wiil permit. If rachitis has developed, **regulation of the diet and of the mode of life** in the manner just indicated is likewise of the utmost importance. After the ninth month of life children may receive, in addition to milk, also eggs, meat-broth, scraped meat, even vegetables, as, for instance, mashed potatoes or scraped carrots. In addition, the patient should receive a saline bath daily (at 35° C. —28° R.—95° F.—with three pounds of salt or six liters of brine) and he should not be permitted to stand too much upon his feet, in order to avoid excessive curvature of the bones. Of drugs, I have prescribed a **powder of iron** and **calcium**:

℞ Iron lactate,
Precipitated calcium phosphate, each, 10.0 (2½ drams);
Magnesium carbonate,
Sodium chlorid,
Sugar, each, 5.0 (75 grains).—M.
Dose: 10 grains thrice daily in milk.

Phosphorus is at present administered by most physicians, and to it a most important influence in the development of bone is attributed; but I have not been able to convince myself that it is capable of any especially favorable result:

℞ Phosphorus, 0.01 (⅙ grain);
Cod-liver oil, 100.0 (3⅓ fluidounces).—M.
Dose: 10 c.c. (1 dessertspoonful) daily.

OSTEOMALACIA.

Etiology.—Osteomalacia is attended with *loss of the calcium-salts on the part of the developed bones,* which therefore become *pliable and soft.* It is doubtful if children, particularly in early life, are attacked by osteomalacia. The disease almost always occurs in *adults*, and in the vast majority of **cases in women.** Etiologically a distinction can be made between *puerperal* and *non-puerperal osteomalacia.* Puerperal osteomalacia develops in conjunction with pregnancy and parturition. It is a noteworthy fact that osteomalacia exhibits a remarkably unequal *geographic distribution.* It occurs with especial frequency along the Rhine, in Switzerland, and in certain portions of upper Italy. The causes for this are not understood. Nothing of a definite nature is known with regard to the etiology of non-puerperal osteomalacia. **Exposure to cold** and **traumatism** have been mentioned as causes. Derangements of the nervous system also appear to be of etiologic influence, as the disorder has in a number of instances been encountered in association with **mental disease** and **exophthalmic goiter.** Osteomalacia is by some clinicians considered a result of **auto-intoxication** depending upon metabolic disturbances in the ovaries; by others it is looked upon as an **infectious disease.**

Symptoms.—Osteomalacia is frequently preceded by drawing **pains** in the bones, joints, and muscles in the form of *premonitory symptoms—prodromes.* The patients tire readily in walking, and eventually are scarcely able to sustain themselves upon their legs. The softening of the bones is readily followed by their **curvature,** and this constitutes one of the most valuable objective symptoms of osteomalacia. In cases of puerperal osteomalacia the *pelvis* suffers first, the promontory of the sacrum projecting far forward into the pelvic cavity, while the acetabular regions are pushed inward, so that the symphysis pubis projects forward like a proboscis and the pelvis acquires the form of a heart. As a result the pelvic diameters become so reduced that insurmountable obstruction to labor results, and even the bladder and the rectum are compressed and their functional activity interfered with. Curvature of the *vertebral column* develops, kyphosis with especial frequency. This is occasionally so pronounced in the cervical region that the chin comes to rest upon the anterior aspect of the chest. The **thorax,** also, often becomes greatly deformed, and the lungs and the heart must accommodate themselves to the new conditions. The patients, therefore, frequently complain of bronchial catarrh, dyspnea, and palpitation of the heart. The **extremities** likewise do not remain free from deformity. They readily undergo infraction, and not rarely a slight fall may give rise to fracture of various bones. The body occasionally shrinks to dwarf-like proportions, particularly if the curvature of the spine be marked. **Febrile movement** is not rarely present. In a number of instances an increased amount of lime has been demonstrated in the **feces,** but no constant alterations in the **urine** are known to occur. Increased elimination of calcium and phosphoric [illegible] lactic acid, nucleo-albumin, and hemi-albumose has been [illegible] some cases of osteomalacia. The urine often contains a precipitate of phosphates and carbonates, which may give rise to the formation of calculi. Occasionally **nervous disturbances** appear, such as muscular wasting, tremor, spasm, exaggerated tendon-reflexes, and neuritic symptoms.

Osteomalacia almost always pursues a *chronic course*, extending at least over several months, but generally over several years. Often remissions and exacerbations occur repeatedly, the latter generally in connection with renewed pregnancy. Death results at times in consequence of progressive exhaustion, while at other times it is the result of bronchial catarrh, pneumonia, or weakness of the heart-muscle.

Diagnosis.—The recognition of osteomalacia is exceedingly difficult at the commencement of the disease, and it is possible with certainty only when deformity of the bones has developed. Even then, however, confusion with multiple osteomyelomata is scarcely to be avoided. The latter disease is still rarer than osteomalacia.

Prognosis.—The prognosis of osteomalacia is grave, because permanent recovery rarely takes place. Women are exposed to dangers of especial magnitude in the act of parturition in consequence of pelvic contraction.

Anatomic Alterations.—The bones have occasionally become so soft that they yield and can be cut with a knife, like artificially decalcified bones, or, at any rate, can be readily broken. At times the long bones are so thin as to have acquired an intestine-like character. The bones present abnormal dilatation of the medullary and Haversian canals. In the spongy bones the osseous trabeculæ are often absent, so that large spaces filled with bone-marrow result. The bone-marrow in the early stages appears red and rich in blood, but subsequently it acquires a rather yellowish, then a grayish, color, and it undergoes atrophy.

On *microscopic examination of the bones* the marked dilatation of the medullary spaces and the Haversian canals is likewise conspicuous. In the immediate neighborhood the osseous tissue has lost its calcium-salts, stains therefore with carmine, and exhibits a striated appearance. Often the bone-corpuscles have undergone degeneration. The medullary spaces and the Haversian canals exhibit numerous diverticula in the bones (Howship's lacunæ) in which myeloplaques are situated. The bones are unusually light, and contain little inorganic matter, particularly lime-salts. The bone-fluid is said occasionally to possess an acid reaction. The presence of lactic acid in the bones has also been reported, and this is thought to effect solution of lime-salts. Other physicians, it is true, attribute the solvent action to carbon dioxid, which probably accumulates in excessive amount in the vascular medullary tissue.

Treatment.—The treatment is similar to that for rachitis (p. 305). I have as yet been unable to convince myself with certainty of the usefulness of phosphorus. The patients should be admonished of the danger of falling, and women, particularly, of that of renewed pregnancy. Recently both ovaries have been removed in desperate cases—thus *castration* performed.

DEFORMING ARTHRITIS.

Etiology.—Deforming arthritis generally avoids early life, and develops as a rule *after the fortieth year*. The disease occurs most frequently in the poorer classes of the community, so that it has been designated also the *arthritis of the poor*. Among the causative factors **exposure to cold** is mentioned, emphasis being placed upon the fact that moldy and moist dwellings and working in the damp favor the development of the disorder. An unfavorable influence is ascribed, also, to **traumatism.** Occasionally the alterations in the joints develop after antecedent **infectious diseases.** Finally certain **diseases of the nervous system** are believed to be of etiologic significance, particularly hysteria and chronic diseases of the spinal cord.

Symptoms.—The disease generally *sets in insidiously*, and involves at times but a single joint or many joints, in the latter event often exhibiting a strikingly symmetric distribution. The *monarticular variety of deforming arthritis* occurs with especial frequency in the hip-joint, is attended with pain, stiffness, and, finally, immobility. The condition is known as senile disease of the hip-joint, and is much feared. *Polyarticular deforming arthritis* involves with preference either the large or the small joints, although simultaneous involvement of the most varied joints is not uncommon. The patients complain generally at first of **pain,** which is not confined alone to the affected joint, but extends throughout an entire extremity, and is frequently aggravated by change in the weather and after exposure to cold. The pain is increased on pressure and on movement. The joints gradually become the seat of **enlargements** of bony hardness and of **deformities,** which render movement progressively more difficult, and eventually result in ankylosis. Movement of the joints is not rarely attended with palpable and audible **grating** and **crackling.** These alterations are unattended with fever and symptoms of local inflammation in the joints. Should numerous joints be involved the patients become rigid, immobile, and helpless, and are forced to seek the aid of others. Peculiar distortion and deformities occur in the **fingers** and the **toes.** In the hands, the thumb and the little finger often are uninvolved, while the remaining fingers become markedly flexed upon the palm at the interphalangeal joints, and luxation toward the ulna occurs in the metacarpophalangeal joints, so that the fingers are superimposed upon one another like the shingles of a roof (Fig. 54). A number of other displacements of the fingers also occur. Often small, hard nodules —so-called *Heberden's nodes*—are encountered between the middle and the terminal phalanges. If the **vertebral column** is the seat of arthritis deformans, it often becomes rigid and immobile, and deformity and stenosis of the vertebral canal may result in compression of the spinal cord, with spinal pressure-paralysis.

The disorder pursues a *chronic course*, and not rarely extends over many years. It is not attended with endocarditis, but on the other hand marked *arteriosclerosis* often develops. *Nervous disturbances* also not rarely occur, particularly paresthesia, muscular contractures, muscular wasting, and hemicrania. At times *amyloid degeneration* has been observed. Death results in consequence of progressive marasmus or intercurrent disease.

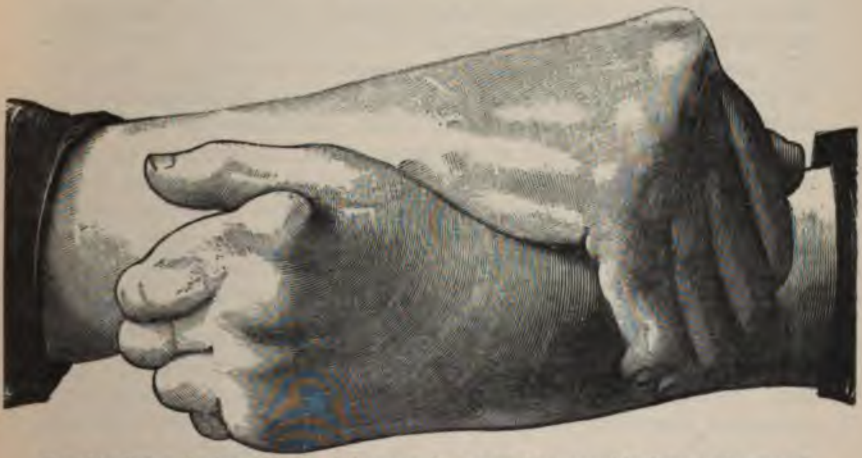


FIG. 54.—Polyarticular arthritis deformans in a man, 52 years old; from a photograph (personal observation, Zurich clinic).

Diagnosis.—The recognition of deforming arthritis is unattended with difficulty. The disorder is distinguished from **chronic articular rheumatism** with secondary deformity of the joints by the absence of febrile attacks and signs of local arthritis (swelling in consequence of exudation, redness, and cutaneous edema). In cases of **uric-acid arthritis** gouty nodules should be looked for, particularly in the ears.

Prognosis.—The prognosis is unfavorable in so far as it is impossible to prevent with certainty the progress of the disease and to effect involution of articular deformity, although life at first is not immediately threatened.

Anatomic Alterations.—In conformity with the symptoms during life, the affected joints when opened exhibit more or less marked deformity. Synovial fluid often is wanting, and intra-articular adhesions are not rarely present. The articular cartilages, particularly at the central portions of the joint, are destroyed, while the peripheral portions exhibit hyperplasia, in which, also, the articular extremities of the bones are involved. The articular capsule often is the seat of thickening, and even of calcification, and its inner surface presents hyperplasia of the articular villi, which likewise are often in part ossified. Occasionally some villi have become detached, and lie in the articular cavity as free bodies.

Treatment.—**Potassium iodid** (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily) especially has been recommended in the treatment of deforming arthritis. Salicylic acid, sodium salicylate, salol, salophen, salipyrin, antipyrin, and phenacetin usually afford little relief to the articular pain. **Massage of the joints with salicylated ointment** (salicylic acid, 10.0—2½ drams; wool-fat and lard, each, 25.0—¾ ounce) and **enveloping the joints in salicylated cotton** may be recommended. The symptoms are usually mitigated also by means of **hot baths.** Well-to-do patients may be sent during the summer to *indifferent thermal baths* or to *sulphur-baths, saline baths, peat-baths,* or *mud-baths.* **Sand-baths** and the **local employment of hot air** are also in good repute. Nothing can be accomplished with **electricity** (local application, galvanization of the sympathetic). The patient should be guarded against exposure to cold and wetting of the body, should always avoid damp dwelling-rooms, and should wear flannel clothing next to the skin. Those who are favorably situated will do well to spend the cold and changeable period of the year in a uniformly warm climate, as, for instance, on the Riviera or, still better, in Egypt.

PART X.

INFECTIOUS DISEASES.

INFECTIOUS DISEASES OF TYPICAL LOCALIZATION.

I. ACUTE INFECTIOUS EXANTHEMATA.

TYPHUS FEVER.

Etiology.—Typhus fever is a highly *contagious disease*, to which nurses and physicians in the pursuit of their avocation have often fallen victim. The infective material is not known, but it is believed to be some form of lower organism. Infection may take place by *transmission through the air*, through *personal association*, through the *intermediation of a third person*, and through *articles in common employ*. Typhus fever still occurs in *endemic distribution* in Ireland, Russia, Galicia, Hungary, and Italy. The disease is often conveyed from these countries through emigrants, peddlers, laborers, and vagrants, and it has repeatedly been possible to demonstrate that it has appeared in all places that have been visited in their travels by individuals infected at home. Only recently typhus fever was conveyed to cities in Northern Germany, in which extensive epidemics occurred. The circumstance is especially favorable for the transmission of the disease that infected persons are infective at a period when they feel tolerably well and are capable of travelling. All places in which they sleep over night are constituted sources of infection for those who follow them, and accordingly experience demonstrates that epidemics of typhus fever frequently originate from certain *lodging-houses* of low and uncleanly character. *Police-stations* and *prisoners* also are not rarely infected through vagrants, and from these sources the disease may be widely disseminated. In earlier times typhus fever was more frequently encountered than at the present day, because little importance was attached to measures of isolation and disinfection. The disorder occurred with especial frequency in times of war and of famine, whence it was designated also *war-typhus* and *famine-typhus*. It is comprehensible that the intimate association of many persons in times of war is especially favorable to the spread of the disease. An individual who has recovered from an attack of typhus fever is generally exempt from subsequent attack in the future, because an *acquired immunity* results. In isolated instances typhus fever has been observed in *association with some other infectious disease*, as, for instance, smallpox or malaria.

Symptoms and Prognosis.—Typhus fever pursues the course of an **acute infectious disease.** It generally sets in with a single severe chill, which is followed by continued fever that terminates suddenly at the close of the second or the third week of the disease amid critical symptoms. Infection is not immediately followed by the development of morbid manifestations; but, on the contrary, the infected individual remains apparently healthy for from one to three weeks before the first symptoms are manifested. This interval is designated the *period of incubation*.

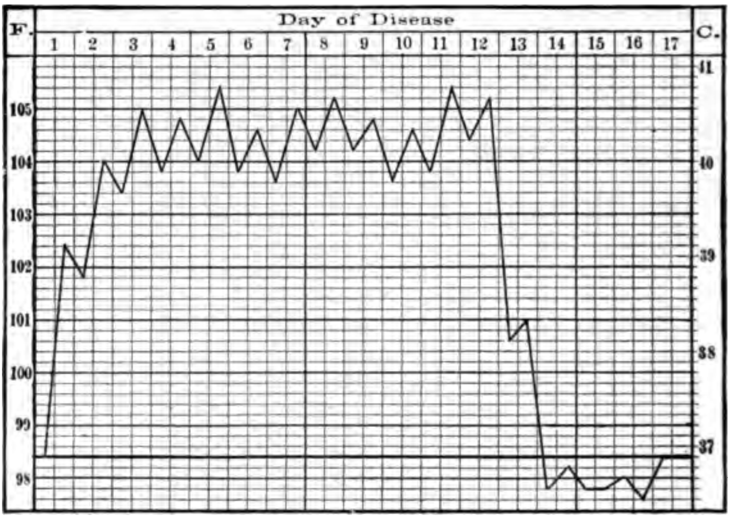


FIG. 55.—Temperature-curve in a case of typhus fever (Salinger and Kalteyer).

The period of incubation is followed by the *stage of premonitory symptoms*, or the *period of prodromes*, which is manifested by febrile general infection. A chill suddenly occurs, which is followed by fever and elevation of temperature to 39° or 40° C. (102.2° or 104° F.). With this are associated an appearance of redness, acceleration of pulse and of respiration, loss of appetite, increased thirst, and evacuation of a small amount of red (high-colored, concentrated) urine. The patient generally complains of headache and vertigo, is no longer able to sustain himself upon his legs, and often soon falls into a state of unconsciousness and delirium. Examination of the internal viscera frequently reveals the presence of bronchial catarrh, and the discovery of *enlargement of the spleen* particularly is important. The liver also is often enlarged. The prodromal period may last *from three to five days*, and then is followed by the *stage of eruption*.

The *stage of eruption or exanthem* is characterized by the appearance of a cutaneous rash. This occurs in the form of roundish, bluish-red, ill-defined spots, on the average as large as a pea, and fading on pressure, which are dependent upon circumscribed hyperemia of the skin and are nothing more than *roseolæ*. The roseolæ generally appear first at the junction of the chest and the abdomen, whence they extend over the entire trunk, the arms, and the legs, and eventually they may appear upon the face, in this situation with especial distinctness in children and in individuals with a delicate skin. Occasionally the body is covered with thousands of such spots. The individual spots persist for about ten days, and then disappear, with slight desquamation of the skin. At times a number of fresh spots develop even during convalescence. Not rarely *petechiæ* in greater or smaller number appear, together with the roseolæ. During the stage of eruption the fever persists in an unchanged manner. Occasionally transitory depression of temperature occurs toward the end of the first week of the disease, but this soon disappears, and becomes permanent only toward the close of the second or the beginning of the third week.

The *crisis* generally begins with profuse perspiration, in the course of which, within from six to twelve hours, complete defervescence occurs. At times the crisis is immediately preceded by increased elevation of temperature, active delirium, and even a chill, phenomena that are without serious significance and are designated *critical perturbation*. The rapidity with which patients recover is often surprising. *Relapses* occur but rarely.

The *prognosis* of typhus fever is always serious, as in some epidemics death has occurred in more than half of those attacked. In the *aged*, in *drunkards*, and in those suffering from *disease of the heart* the prognosis is especially grave, because such individuals resist poorly all febrile infectious states and readily die in consequence of paralysis of the heart. Pregnant women also are exposed to greater danger; abortion and puerperal septicemia frequently occur in them. Persistent temperature above 42° C. (107.6° F.), and acceleration of the pulse beyond 120, are likewise serious indications. *Paralysis of the heart*, probably the result rather of intoxication with bacterial poisons (toxins) than of the high fever, is an important danger in every case of typhus fever. It may occur not only at the height of the disease, but also not rarely during the crisis, and even during convalescence. In other patients *complications* become a source of danger. Certain inflammatory complications, such as pneumonia, are of especial gravity, while others are of less significance. Complications that occur with especial frequency in cases of typhoid fever, and thereby give the disease a peculiar impress, are scarcely known. Complications are generally due to secondary infection with pyogenic bacteria, most frequently the Streptococcus pyogenes; and whether one or another organ affords entrance to the excitants of inflammation and the conditions for proliferation, and responds with inflammation, is dependent upon factors that are largely unknown. *Sequelæ* may occasionally result from complications, but of these none is distinctive of typhus fever.

Diagnosis.—The recognition of typhus fever is generally easy. The disease is differentiated from **measles** by the fact that the latter is distinctively a disease of childhood, that the spots of measles appear earliest upon the face, and that measles generally pursues a favorable course. In cases of **syphilis** the fever is generally slight, and, besides, other evidences of syphilis will generally be present at other parts of the body, particularly the genitalia and the pharynx. In cases of **typhoid fever** the Widal blood-serum reaction is decisive. Besides, roseolæ are but rarely found, and in any event only isolated, upon the extremities in cases of typhoid fever, but never on the face. Typhoid fever also is generally attended with diarrhea. Further, the pulse is slow as compared with the temperature. The search for typhoid-bacilli in the urine, the feces, and the blood from the roseolæ will generally be too time-consuming for the practitioner.

Anatomic Alterations.—As the distinctive cutaneous eruption is generally no longer appreciable in the dead body, the anatomic findings generally are not sufficient for the subsequent recognition of typhus fever. The body exhibits only such changes as attend any febrile infectious disease. The **muscles of the trunk and extremities** are dry, of dark-red, ham-like color, and occasionally are the seat of hemorrhages and necrotic areas of softening, particularly in the abdominal muscles. Microscopic examination discloses granular clouding and fatty and waxy degeneration of the muscular fibers. Similar alterations occur also in the **myocardium**, which, at the same time, is characterized by great flabbiness. Granular clouding and fatty degeneration are present also in all **glandular cells** (liver-cells, epithelial cells of the convoluted uriniferous tubules, the glands of the stomach, the intestine, the pancreas, and the salivary glands). The **spleen** is increased in size, and generally presents a soft, almost diffluent consistence.

Treatment.—No *specific remedy* for typhus fever is known. Recently **blood-serum therapy** has been recommended as a specific form of treatment, the blood-serum of convalescents from typhus fever being injected subcutaneously into persons still at the height of the disease. The fever is said to have declined as a result, and the disease to have pursued a more rapid and a more favorable course. This method of treatment has, however, not yet received extensive practical application. Accordingly one must content himself with **expectant treatment.** The patient accordingly receives a liquid diet, consisting principally of milk, while thirst is quenched with some good spring-water, to which, in accordance with individual taste, an equal amount of white or red wine is added, and between eight and ten in the morning, and between four and six in the afternoon, a tepid bath at 35° C. (28° R.—95° F.) is taken, in which the patient remains from fifteen to twenty minutes. After the bath he is vigorously rubbed with warm towels; also the shirt and the bed should be warmed. The patient is refreshed by the bath, which is therefore agreeable to him and it prevents gangrene from pressure upon the skin. If the patient should be comatose, care must be taken to change the position of his body every hour, in order to prevent pressure-necrosis of the skin. The sheets should be perfectly smooth, and dried bread-crumbs especially should not be permitted to remain in the bed. Also over-distention of the bladder should be guarded against, as this may give rise to uremia. Something to drink should be offered the patient every half-hour. It is important to secure cleanliness of the mouth in order to prevent infection and suppuration of the salivary glands. This is best effected by irrigation of the mouth with potassium chlorate (5.0 : 200—75 grains : 6½ fluidounces) after each of the three principal meals. Internal remedies are not necessary; should they be demanded by the patient, acids may be administered, which relieve the sense of thirst in some degree, as, for instance:

℞ Solution of phosphoric acid, 5.0 : 180 (75 grains : 5½ fluidounces);
Sirup of raspberry, 20.0 (5 fluidrams).—M.
Dose: 15 c.c. (1 tablespoonful) every two hours.

Symptomatic treatment will be necessary when individual symptoms become exceedingly troublesome to the patient, or acquire an intensity that is dangerous to life. For the relief of distressing headache and stupor an *ice-bag* should be prescribed. If the *fever* be persistently high (above 41° C.—105.8° F.), or in the case of the aged, of drunkards, in the presence of heart-disease, and in pregnant women, who, experience has shown, bear fever badly, *antipyretics* may be administered, even when the temperature is slightly above 39° C. (102.2° F.), as, for instance, phenacetin 1.0 (15 grains). Should the fever prove resistant to drugs, the patient should be kept *continuously in a warm bath* at a temperature of 37° C. (29.6° R.—98.6° F.) until the temperature has been reduced and remains low. In the presence of **threatening collapse** *stimulants* should be employed (cognac, champagne, mulled wine, egg-nog, camphorated oil subcutaneously, etc.).

In the management of the disease great importance is to be attached to **preventive measures—prophylaxis.** Patients suffering from typhus fever should be treated in isolation-hospitals, as admission to the general wards of a hospital would expose other patients to the danger of infection. The patients should have special nurses, and, if possible, also special physicians. In any event, physicians should visit cases of typhus fever last, and should disinfect themselves after each visit. The transmission of letters, and of articles generally, is to be interdicted. Should the patient remain at home the house should be officially quarantined. The rooms and utensils, the linen, and the clothing of the patient should be carefully disinfected; articles of little value, such as sacks of straw that have served as mattresses, are best burned. The walls of the room should be rubbed down with bread-crumbs, the floor, the ceiling, and the furniture washed or wiped with mercuric chlorid, and the linen and clothing sterilized by live steam in a disinfecting-apparatus. Boarding-houses and jails in which cases of typhus fever have resided are best closed for some time. After disinfection has been accomplished, it is advisable to keep the windows and the doors of the rooms open for days. In case of death the *funeral* should be strictly *private.* Exposure of the body and a public ceremony should be forbidden on account of the danger of infection. It is especially important to apply rigidly the prophylactic measures mentioned in all places where typhus fever prevails epidemically, in order to eradicate the disease and to render impossible its dissemination elsewhere. Strict quarantine at boundary-lines and the suppression of vagrancy will also contribute to the same end.

MEASLES (MORBILLI).

Etiology.—Measles occupies a prominent place among the *acute infectious diseases of childhood*—not that children possess an especial predisposition for the virus of measles, but that as a result of social conditions exposure to infection with measles generally takes place during childhood, and the disease is thus acquired. Should adults, exceptionally, be attacked by measles, they will generally prove to be individuals who have led a secluded life in youth or have quite accidentally escaped exposure to infection. *Congenital immunity* to measles occurs with extreme rarity, and some individuals may escape the disease throughout life, although frequently exposed to the risk of infection. *Immunity is acquired* by those who have recovered from an attack of measles; the same individual is but rarely attacked by measles twice or even thrice in the course of his life.

The *virus of measles* has not yet been isolated, and all of the reports with regard to the finding of cocci, bacilli, and protozoa are as yet without the necessary confirmation. *Inoculation-experiments* on human beings have shown that the virus is contained in the blood, the tears, the nasal, the laryngeal, and the bronchial secretion, as well as in the contents of cutaneous vesicles that may be present. On the other hand, inoculation-experiments with scales of skin have invariably failed. Infection may take place through the *air*, through *immediate contact with a case of measles*, through the *intermediation of a third person*, or through *inanimate objects*. Children acquire measles most frequently in school and on public playgrounds where they associate with children apparently suffering from only nasal or bronchial catarrh, although these symptoms are really the commencement of the measles.

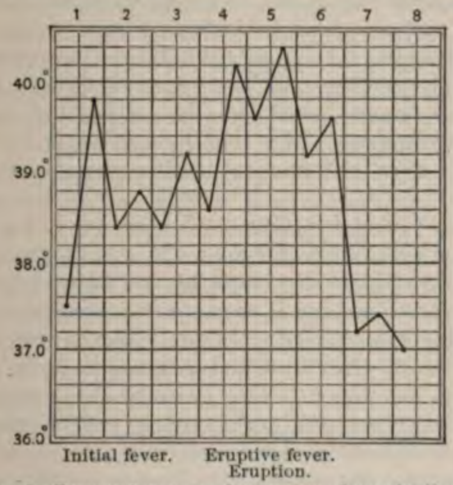


FIG. 56.—Temperature-curve from a case of measles (Anders).

Measles frequently occurs in *epidemic*, and occasionally in *pandemic*, distribution, and in the latter event the disease may be distributed over extensive areas of land. *Sporadic cases of measles* occur not at all rarely in large cities, and they constitute the foci of origin for epidemics, which, when the patients travel, may also appear suddenly in previously healthy cities. Experience has shown that in some large cities epidemics of measles recur from every two to every six years with a certain degree of regularity. They occur more frequently in the winter and spring than in the uniformly warm summer or in the autumn, and they continue on an average for from four to six months. Occasionally other epidemic diseases, particularly whooping-cough and chicken-pox, precede epidemics of measles, or follow them, or exist simultaneously.

Symptoms.—The *period of incubation* of measles averages ten days. Toward the end of this period the children not rarely exhibit pallor, loss of appetite, disturbed sleep, capriciousness, and a lacrimose tendency. Occasionally slight febrile movement also occurs. The *prodromal* or *exanthematic stage* frequently sets in with a chill, or repeated slight chilliness, followed by **fever,** with elevation of temperature to 39° or 40° C. (102.2° or 104° F.). At the same time **inflammatory symptoms** become manifest upon various **mucous membranes,** representing nothing more than the eruption of measles. The ocular conjunctiva becomes reddened and swollen, and the eyes are the seat of burning and dryness, and of a sense as if a foreign body were present. The eyes become sensitive to light, and the secretion of tears is increased. A feeling of burning and tickling dryness in the nose becomes appreciable; frequent sneezing, increased secretion from the mucous membrane, and occlusion of the nares result. The pharyngeal and laryngeal mucous membrane exhibits punctate or diffuse redness, is swollen, and secretes excessively. At the same time the patient complains of pain in swallowing and of cough, and is not rarely hoarse. The exanthematic stage generally continues for *three days,* the fever generally growing less from day to day.

With the onset of the *exanthematic stage* the temperature again rises, and generally persists for four days at a continuous level, then suddenly to decline by *crisis* to the normal level. A distinctive feature of this stage is the occurrence of the **exanthem of measles** upon the external integument. The eruption consists of *roseolæ,* therefore of circumscribed cutaneous hyperemia, fading on pressure with the finger. The spots are of roundish form, on an average as large as a pea, and in many parts of the body they are discrete and distinctly separated from one another, while in other situations they are confluent. The first spots appear upon the face, particularly on the forehead, the cheeks, and the chin; but they can be found, also, upon the scalp.[1] Within from twenty-four to thirty-six hours the trunk and the extremities also are covered by the spots. They may be found in large number even upon the palms of the hands and the soles of the feet. At times the patients complain of a sense of **prickling** and of **slight burning in the skin.** Also, the **peripheral lymphatic glands** are frequently enlarged and sensitive to pressure. The exanthem generally reaches the height of its development on the third day, and this is known also as the *acme* or *stage of efflorescence;* but then the eruption begins to fade progressively, and earliest from those portions of the body on which the first spots appeared, namely, the face, and, latest, from the extremities.

---

[1] The eruption may usually be detected in the throat a day or two in advance of its appearance on the skin. Small, round, bluish-white spots, surrounded by an area of congestion or upon a diffuse red background, may be seen on the mucous membrane of the mouth, especially opposite the lower molar teeth. Sometimes a sense of rubbing or crackling can be elicited on pressure upon the abdomen from the presence of the eruption on the peritoneum.—A. A. E.

The stage of exanthem is followed by that of *desquamation.* Numerous small scales of epidermis form upon the skin, again first on the face, and these are exfoliated within a week, so that the patients are well again at the end of the fourth week of the disease following infection. As the patient is now free from fever, it is generally difficult to keep him in bed and to protect him against indiscretions in spite of the desquamation. *Relapses of measles* occur but rarely. Occasionally measles exhibits deviations from the course described—*anomalies*—of which the principal may be mentioned. At times these are related to indifferent *variations in the exanthem.* Thus, the spots of measles may be papular or vesicular, or be intermixed with hemorrhages into the skin. *Measles without exanthem* is yet worthy of mention, an eruption upon the external integument being wanting. *Measles without enanthem* also occurs. Under other anomalous conditions associated with measles the *general infection* exhibits an unusual character. Occasionally elevation of temperature fails to take place—*afebrile measles;* or the general infection proves to be of especial severity, either because the temperature is unusually high and persistent, or because signs of rapid and profound exhaustion make their appearance. Cases of the latter variety are designated also *adynamic measles.* At times symptoms of blood-dissolution appear. Under such circumstances extensive hemorrhages, difficult of control, appear upon the skin and the various mucous membranes, whence the name *hemorrhagic measles,* and to which occasionally the patients quickly succumb.

Complications also may be attended with serious dangers, some of which are peculiar to measles by reason of their frequency of occurrence. While complications are rare in some epidemics of measles, they occur with extraordinary frequency in others, and thereby stamp the disease as of serious character. It will suffice in the following to mention only the more common complications, as eventually any organ may be the seat of inflammatory complications in the course of measles. During the exanthematic and the enanthematic stage, occasionally, however, not before the commencement of the stage of desquamation, the *swelling of the laryngeal mucous membrane* at times becomes so excessive that the patient is seized with marked dyspnea. This occasionally occurs in paroxysms, particularly during the night, and simulates pseudocroup (p. 81, Vol. I.). The conditions are far more serious if *fibrinous inflammation of the laryngeal mucous membrane* develops—thus *laryngeal croup*—which often causes death by suffocation within a few hours. *Bronchial catarrh* is almost constantly an accompanying manifestation of measles, and in itself without serious significance. It becomes attended with danger when it extends to the pulmonary alveoli, and is associated with *bronchopneumonia*, which has been designated also simply *measles-pneumonia.* It thus appears that the respiratory organs especially furnish the basis for serious complications in cases of measles. The heart, the stomach, and the intestines are less commonly involved, but at times diarrhea of such an intractable character occurs as to lead to a fatal termination. The occurrence of *otitis media* further is worthy of mention, as it not rarely gives rise to serious cerebral complications (sinus-phlebitis, meningitis, cerebral abscess) or to loss of the sense of hearing. Should the inflammation involve both the ears, complete deafness may result, which, if it occurs in children who have not yet learned to speak, may be attended with mutism.

Measles but rarely occurs *simultaneously with other infectious diseases* in the same person, as, for instance, whooping-cough, small-pox, chicken-pox, mumps, typhoid fever.

Among the *sequelæ* of measles there may be mentioned, in addition to *loss of hearing* and *deaf-mutism, chronic laryngeal catarrh,* which for a long time may be followed by hoarseness, occasionally, also, by dyspnea, and these may be intensified paroxysmally in an alarming degree. *Bronchial catarrh* also persists at times for long periods. Occasionally bronchopneumonia is followed by *pulmonary tuberculosis* of caseous or miliary variety, to which the patient succumbs. I have in a number of instances observed death occur in vigorous individuals between the fifteenth and the twenty-fifth year of life from miliary tuberculosis, and especially from tuberculous meningitis, originating from *tuberculous-caseous bronchial glands,* the latter, from the history, being apparently developed in early childhood during an attack of measles. Measles is often followed by *scrofulosis,* as manifested by enlarged lymphatic glands and a tendency to chronic inflammation of the skin, the bones, and the joints.

Anatomic Alterations.—The anatomic alterations in the skin must be studied in portions of integument removed during life, as some of the manifestations will have disappeared in the dead body. In the situation of the spots of measles the blood-vessels of the cutis are naturally found dilated. Accumulations of round cells in small number also are present, partly in the immediate vicinity of the blood-vessels, and partly adjacent to the hair-follicles and the sebaceous follicles and between the erectors of the hair.

Diagnosis.—The recognition of measles is easy. In contradistinction from **Rotheln,** the more marked febrile general infection particularly is distinctive of measles. **Syphilitic roseola** occurs generally in adults, and, in addition, other symptoms of syphilis will be present, particularly on the genitalia and in the pharynx.

Drug-exanthems, such as develop after the use of salicylic acid, antipyrin, quinin, oil of turpentine, balsam of copaiba, and others, are often unattended with fever and involvement of the mucous membranes. In addition, the history should be taken into consideration. **Typhus fever** is attended with more profound general infection, the involvement of the mucous membranes is inconspicuous, and the spleen is enlarged. Measles cannot be differentiated with certainty from **small-pox** during the first twenty-four hours of the eruption, but after this time papules begin to replace the measles-like spots upon the skin in cases of small-pox, and soon, also, vesicles and pustules. It is noteworthy that in cases of small-pox severe sacral pain is frequently present during the prodromal period. **Scarlet fever,** in contradistinction from measles, is attended with diffuse redness of the skin. Should this occur in places also in cases of measles, distinctly circumscribed formation of spots will be found in other situations. Vomiting frequently occurs during the prodromal period of scarlet fever. Inflammation of the pharynx also is a prominent symptom of scarlet fever. The red tongue of scarlet fever further is noteworthy. All possible diagnostic difficulties may arise particularly in the presence of sporadic cases of measles, or if an epidemic of Rötheln, scarlet fever, small-pox, or typhus fever prevails at the same time as an epidemic of measles.

Prognosis.—Measles is a benign disease from which most persons recover without bad effect. Dangers result principally from complications, by the frequency of which some epidemics are, in fact, characterized.

Treatment.—There is as yet no **specific treatment** for measles. Blood-serum therapy has been employed in isolated instances, and favorable results have been recorded. In the majority of cases **expectant treatment** will suffice. On account of the photophobia the **sick-room** should be darkened, and the head of the patient be placed toward the window. A tepid bath (35° C.—28° R. —95° F.) should be given night and morning, and the body be subsequently anointed with a **carbolated ointment** (carboli acid, 5.0—75 grains; wool-fat and lard, each 25.0—$\frac{3}{4}$ ounce) which yields a pleasant sense of coolness and disinfects the skin **Irrigation of the mouth** and **gargling** with a solution of potassiu chlorate (5.0 : 200) should be practised after each meal, in orde to avert inflammation in the oropharyngeal cavity and its exte sion to the tubes and the excretory ducts of the salivary gland The *food* should be liquid, and consist principally of milk a meat-broth. Should serious symptoms and complications ari these should be attacked in the usual manner, and expectant placed by symptomatic treatment.

In order to prevent the spread of measles **preventive measure** **prophylaxis**—will be necessary similar to those that have been m tioned on p. 316 for typhus fever, but they will be resorted to only when an epidemic of measles has acquired dangerous characters, or if those exposed to the risk of infection are greatly debilitated or otherwise ill. As almost all persons are attacked once by measles, it is, under favorable conditions, not undesirable for children to acquire the infection, in order thereby to be protected against the disease for the future.[1] Measles probably is already infective during the period of incubation. It may be transmitted also through the intermediation of a third person; so that all the children of a family in which measles is present should be excluded from attendance at school. The conscientious physician will visit his cases of measles last.

RÖTHELN (RUBEOLA).

Etiology.—Infection with Rötheln takes place in the same way as infection with measles. This occurs most frequently in *schools* and *public playgrounds*, through intimate association with those already infected and diseased. Less commonly, conveyance of the as yet wholly unknown virus takes place through the air, or through the intermediation of a third person or of inanimate objects.

Rötheln is pre-eminently a *disease of childhood*, occurring generally in epidemic distribution, but much less rarely than measles. Many physicians do not consider Rötheln an independent infectious disease at all, but merely a mild variety of measles. I cannot agree with this view on the basis of personal experience, and this position is taken by almost all physicians who are familiar with Rötheln from personal observation.

Symptoms, Prognosis, and Diagnosis.—The *period of incubation* is said to be from fourteen to twenty-one days. *Prodromes* are wanting, or they consist in general malaise, pallor, loss of appetite, and disturbed sleep. Occasionally slight febrile movement occurs, together with conjunctivitis, rhinitis, pharyngitis, and laryngitis, the inflammation of the mucous membranes mentioned representing an enanthem. The *stage of eruption* likewise is occasionally unattended with fever. In any event, such elevation of temperature as may be present does not attain any considerable height. There appear at first upon the face and the neck, then upon the trunk and extremities, reddish, circular spots, on an average about as large as a pea, resulting from circumscribed hyperemia of the skin, therefore, roseolæ. These often disappear in the course of a few hours, so that generally only some portions of the body are covered with spots, and not the entire body. Often the spots are quite pale red. The patients complain at times of slight **itching of the skin**, and exhibit slight **enlargement of the lymphatic glands.** Frequently the disappearance of the spots is followed by slight **desquamation of the skin.** *Complications* (herpes, bronchitis, pneumonia, albuminuria) are rare, as are also *sequelæ*. *Relapses* are but rarely observed. Recovery from one attack of the disease confers acquired *immunity* for the future. The *prognosis* accordingly is favorable. The *diagnosis* is easy in the presence of an epidemic of Rötheln in which all of the cases exhibit approximately similar features. Sporadic cases cannot be differentiated with certainty from mild cases of measles.[1]

---

[1] It may be legitimately objected to such advice, in the first place, that all individuals are not necessarily attacked at some time by measles; and, in the second place, that the complications of the disease and the possibility of a fatal issue are not lightly to be invited, apart from the fact that each case must obviously add to the avenues of infection.—A. A. E.

Treatment.—The treatment is the same as that for measles.

SCARLET FEVER.

Etiology.—Like measles and Rötheln, scarlet fever also is one of the *infectious diseases of childhood;* nevertheless, it occurs likewise in adults, and much more commonly than measles. The *tendency* (*predisposition*) to scarlet fever is less widely disseminated than that to measles, and many individuals escape the disease throughout life. A considerable number of persons thus appear to possess a *congenital* (*natural*) *permanent immunity* to scarlet fever. In some the *immunity* is but *transitory*, so that the individual may escape on one occasion when exposed to infection and be attacked on another occasion. *Acquired immunity* to scarlet fever is generally possessed by those who have recovered from a previous attack, for *repeated attacks* of the disease are rare. Under certain conditions an *exaggerated predisposition to infection with scarlet fever* is appreciable. This is exhibited in cases of *traumatic scarlet fever*, which develops in conjunction with traumatism and begins at the seat of injury. The *scarlet fever of parturient women* probably is scarcely other than a special variety of traumatic scarlet fever. Further, the diagnosis of traumatic scarlet fever must be made with care, because septic exanthems may closely simulate scarlet fever.

The *virus of scarlet fever* is as yet unknown. Inoculation-experiments in human beings have shown that it must be contained in the blood, the tears, the nasal, the laryngeal, and the bronchial secretion, the urine, perhaps also the fæces, and in the scales of skin. It is present likewise in the contents of such miliary vesicles as may be present. Experience has shown that its vitality and its powers of resistance are quite considerable, as the articles used by scarlet-fever patients have remained infective for more than ten years.

---

[1] It has been suggested that the designation Rötheln comprehends, clinically, two diseases, one presenting the symptoms described, the other being attended with the early appearance of a diffuse, slightly elevated, bright, rosy-red rash over the surface of the entire body, with redness and swelling of the fauces, injection and suffusion of the conjunctiva, enlargement, induration, and tenderness of the lymphatic glands universally, and variable desquamation. The character of the disease is usually mild and the duration brief.—A. A. E.

Infection takes place most frequently through immediate contact and *association with scarlet-fever patients in schools* and in *public playgrounds,* for at the beginning of the disease many patients associate freely with others. Occasionally scarlet fever is conveyed through the intermediation of a *third person,* as, for instance, through the pupils at school from families in which the disease prevails, through those who visit the sick, or through the carelessness of physicians. Transmission may take place also through *domestic articles;* thus epidemics of scarlet fever have in a number of instances been observed among the customers of a given milkman whose children had suffered from scarlet fever. Further, letters, books, unsterilized clothing, and the like, may be the means of transmitting the infection. Infection through the *air* also is possible for a short distance. It will not seldom be learned that it is not possible to demonstrate the source of infection in every case. It may be mentioned, further, that scarlet fever is infectious in all stages except the period of incubation, but probably in the eruptive stage particularly.

Isolated (sporadic) cases of scarlet fever occur frequently in large cities. From them *epidemics of scarlet fever* now and then arise. Occasionally scarlet fever remains confined to a single building with many inmates (orphan-asylums, boarding-houses, barracks), and such *house-epidemics* frequently arise from an imported case of scarlet fever. *Epidemics of scarlet fever* occur most frequently during the cool season of the year, particularly in the autumn months. They generally last longer than epidemics of measles, and occasionally persist for many months in the same place, exhibiting numerous exacerbations and remissions. At times they coexist with epidemics of measles, whooping-cough, and chicken-pox, or they may succeed such epidemics or shortly precede them. At times scarlet fever has been observed in the same patient in association with some other infectious disease (whooping-cough, chicken-pox, typhoid fever).

Symptoms and Anatomic Alterations.—The *period of incubation* of scarlet fever is in some patients scarcely more than twenty-four hours. In any event, it is shorter than that of measles and not more than a week. The *prodromes,* likewise, occasionally persist for only a few hours, at most for one or two days. They often set in with repeated chilliness, followed by elevation of temperature to 39° C. (102.2° F.), rarely above. The patient often complains of **pain in swallowing and in the pharynx,** and the pharyngeal mucous membrane and the tonsils are found greatly reddened and slightly swollen. The redness is either uniformly distributed or it is macular or punctate, and represents only an **enanthem** of the pharyngeal mucous membrane. Gastric symptoms, particularly repeated vomiting, occur with remarkable frequency, a circumstance that is of diagnostic significance.

With the advent of the *eruptive stage* the elevation of temperature frequently increases. The distinctive exanthem appears first upon the neck and in the clavicular region, and then extends in the course of from twenty-four to forty-eight hours over the trunk and the extremities. It is but slightly developed upon the face; the chin and the nose especially appear unusually pale in consequence of local vascular spasm. The *eruption of scarlet fever* gives the skin a uniformly distributed, bright-red appearance. On more critical inspection innumerable small, deep-red punctate spots can be readily distinguished. The redness disappears on pressure, and the condition is accordingly one of cutaneous hyperemia. Microscopic examination of freshly excised portions of skin discloses hyperemia and ampullar dilatation of the blood-vessels of the cutis, round-cell accumulation about the vessels and the follicles, swelling of the cells of the cutis and the rete, and isolated round

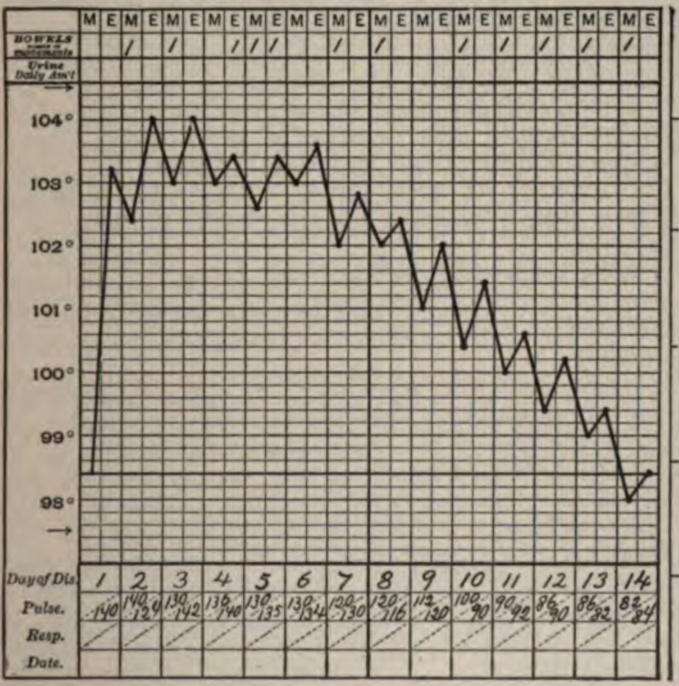


FIG. 57.—Temperature-curve from a case of scarlet fever of favorable course (Anders).

cells among the latter will be found. The turgescence of the skin is increased. The patients complain not rarely of a sense of **burning** and **prickling** or of **itching of the skin,** and frequently also peripheral **lymphatic glands** are swollen, and occasionally even sensitive. The pharyngeal symptoms are often aggravated during the stage of eruption.

The **appearance of the tongue** is quite noteworthy. While at first the tongue is covered with a grayish or whitish coating, this is gradually exfoliated, and an unusually deep-red and clean appearance comes into view. At the same time the fungiform papillæ become enlarged, so that the surface of the tongue exhibits a multinodular aspect. On account of the appearance of the *scarlatinal tongue* it has been designated the *strawberry-tongue,* or the *feline tongue.* The exanthem generally reaches the height of its development on the third or fourth day after its appearance—so-called *acme* or *stage of efflorescence.* After this the eruption gradually fades, and at the close of the week the skin has acquired its usual aspect. The redness disappears earliest from those portions of the skin where the eruption had first appeared.

The **bodily temperature** declines from day to day, and reaches the normal level with the disappearance of the exanthem. Thus, in contradistinction from measles, defervescence occurs not by crisis, but by lysis.

The stage of eruption is followed by that of *desquamation.* At times this immediately succeeds the stage of eruption, even beginning at the close of the latter, while at other times an interval of several days, occasionally of more than two weeks, elapses. Generally the desquamation begins on those portions of the body where the eruption appeared earliest, namely, in the cervical region. The skin under such circumstances looks as if covered with grayish and whitish scales of epidermis, which occasionally resemble bran, so that the skin appears almost as if sprinkled with dust (furfuraceous desquamation). At times, however, it is exfoliated in large coherent membranes (membranaceous or lamellar desquamation); not rarely such desquamation takes place particularly upon the palms of the hands and the soles of the feet. In these situations the skin occasionally peels off like a glove. During the period of desquamation the bodily temperature remains unaltered, and the patients generally feel so well that they can with difficulty be kept in bed. The *duration of the stage of desquamation* is, on an average, fourteen days, so that the patient is convalescent at the close of the fourth week after infection. It is true, cases are not rare in which desquamation continues for a considerable time.

Relapses of scarlet fever are but seldom observed. The *anomalies, complications, and sequels of scarlet fever* are worthy of especial consideration. Among the *anomalies of scarlet fever* cases exhibiting slight *general infection* may be mentioned first. Under some conditions the disease is unattended with fever—*afebrile scarlet fever.* The patient feels well, possibly associates as usual with others, and thereby contributes materially to the spread of the disease. That, further, even such mild cases may be attended with serious complications and sequelæ will be pointed out subsequently. In other instances the general infection is unusually profound. This is exhibited sometimes in the intensity of the fever; at other times in conditions of cardiac weakness or in cerebral disturbances; and at still other times in both sets of phenomena. Occasionally the general infection has become so pronounced already in the prodromal stage that death may result in consequence of hyperpyrexia, or of cardiac or cerebral paralysis. In other instances the symptoms of profound general infection do not appear before the stage of eruption. At times the patient lies in a comatose state, with a distended abdomen, suggesting the clinical picture of typhoid fever or of septicemia, and the designation *adynamic, typhoid,* or *septic scarlet fever* has also been employed. In some cases the profound general infection is indicated by hemorrhages into the skin and the various mucous membranes, to which the patient succumbs—*hemorrhagic scarlet fever.*

Anomalies in the course of scarlet fever are occasionally dependent upon *peculiarities in the character of the eruption*—conditions that are of rather subordinate importance. At times the eruption of scarlet fever does not appear earliest in the cervical region, or it does not involve the entire body, but only some portions thereof—*partial scarlet fever.* Occasionally the reddened skin is covered with small elevations, or with miliary vesicles or larger blebs—*papular, miliary, vesicular,* or *pemphigoid scarlet fever.* Isolated cutaneous hemorrhage in cases of scarlet fever are without significance. *Fugacious scarlet fever* is worthy of note. The redness of the skin persists for only a few hours, and the physician is often in doubt whether the condition is one of scarlet fever or of erythema until the appearance of serious complications of scarlet fever, with particular frequency the scarlatinal nephritis, removes the doubt. *Scarlet fever without eruption* further is of great practical significance. The patient has been exposed to the infection of typhoid fever, is seized with a febrile illness, with symptoms of pharyngitis, occasionally, also, acute nephritis, but a cutaneous eruption is wanting. Nevertheless, cases of infection with well-developed scarlet fever arise from this source. Cases of *scarlet fever without enanthem* also occur, in which the pharynx remains unaffected.

Complications occur with great frequency in cases of scarlet fever, and confer upon the disease its serious characters. They are dependent in part upon the peculiarities of the individual epidemic, and occur with especial frequency in some epidemics.

Under such circumstances it appears that some epidemics are characterized by the especial prevalence of certain definite complications. In the vast majority of cases these are dependent upon *secondary infection with streptococci.* Almost every organ may be involved in inflammation in the course of an attack of scarlet fever, but at this place it will suffice to mention the complications that may be considered typical by reason of their frequent occurrence.

In the first place, the occurrence of necrotic inflammation of the pharyngeal mucous membrane and the tonsils may be mentioned, and which is generally designated *scarlatinal diphtheria.* This appears at times as early as the prodromal stage, but at other times not before the eruptive stage. It is especially attended with grave danger of general septicemia and cardiac paralysis, and some epidemics of scarlet fever are characterized by an extremely high mortality. The condition is generally one of streptococcus-infection of the inflamed tissues, and but rarely infection with diphtheria-bacilli.

An equally serious complication is *scarlatinal nephritis. Febrile albuminuria* occurs quite frequently, and is without serious significance. The conditions, however, are different if the amount of albumin in the urine becomes considerable, if the urine contains tube-casts and blood, and is diminished in amount. Under such circumstances anuria may readily occur, and as a result of this *uremia,* to which the patient not rarely succumbs. In some cases the acute inflammation of the kidneys becomes converted into chronic nephritis, which gradually acquires the characters of secondary contracted kidney, and occasionally terminates fatally through uremia only after the lapse of years. Generally scarlatinal nephritis develops toward the termination of the eruptive stage, or not before the stage of desquamation. Occasionally it is attended with considerable general cutaneous edema. The anatomic alterations are frequently those of acute glomerulonephritis.

Scarlatinal polyarthritis is a much less common complication, and is attended with swelling and pain in the joints. At times purulent arthritis develops, with the danger of septicemia. Streptococci have been demonstrated in the pus in the joints. In the course of polyarthritis, or even independently, *acute endocarditis* sometimes develops, and this may be the source of persistent valvular disease of the heart. Finally, *inflammation of the internal ear* should be borne in mind, occasionally extending to the cerebral sinuses, the meninges, or the brain-tissue, and, as a result, exposing the patient to great danger to life.

Among the *sequels of scarlet fever, chronic nephritis* and *valvular disease of the heart* have already been mentioned. *Stiffness of the joints, chronic otitis,* and *disorders of hearing* may be addi-

Diagnosis.—The recognition of scarlet fever is generally easy. In addition to the distinctive *cutaneous eruption*, the *scarlatinal tongue* and the *inflammation of the pharynx* especially are important in the diagnosis in doubtful cases. Scarlet fever is distinguished from **measles** and **Rötheln** by the fact that it is attended with a diffuse, and not with a macular, redness of the skin. The eruption of measles and of Rötheln may, however, be confluent, and give rise to uniform redness of the skin, but isolated red macules will then always be visible at the periphery of the diffusely reddened skin. Scarlet fever is distinguished from **drug-eruptions** by the fact that the latter occur only after the use of certain medicaments (balsamics, quinin, salicylic acid, antipyrin, potassium iodid, opium, etc.), and that they are unassociated with the scarlatinal tongue and inflammation of the pharynx. The latter circumstance is to be taken into consideration, also, in the differential diagnosis of erythemata generally; besides, erythemata are frequently of transitory character.

Prognosis.—The prognosis of scarlet fever depends essentially upon the severity of the general infection and upon the complications. While some epidemics are attended with scarcely any fatal cases, others are characterized by a frightfully high mortality.

Treatment.—The treatment is the same as that for measles (p. 321). Accordingly, a liquid diet should be prescribed, particularly a **milk-diet**, tepid **baths** at a temperature of 35° C. (95° F.) twice daily, followed by inunctions of the skin with **carbolated ointment** (5 per cent.), and systematic **gargling and irrigation of the mouth** after each meal with potassium chlorate (5.0 : 200). Complications should be treated in the usual manner. **Specific treatment with injections of blood-serum from convalescents from scarlet fever** has thus far not yielded marked success. The results of **injections of streptococcus-serum** are likewise of doubtful character. **Prophylactic measures** should not be neglected: isolation of the patient, preferably in a hospital, sterilization of his clothing, linen, and all personal articles, exclusion from school of the healthy members of the family, disinfection of the living-rooms and the sick-rooms, thorough irrigation of the mouth and nose during convalescence.

ERYSIPELAS.

Etiology.—Erysipelas is a *traumatic infectious disease* resulting from the entrance through wounds into the lymphatic vessels of the skin of the **Streptococcus erysipelatosus**, which at the present time is, not without reason, believed to be none other than the Streptococcus pyogenes.

The contention that in some rare cases other bacteria also, as, for instance, **the** [illegible]**occus** *pyogenes aureus*, and the *typhoid-bacillus*, in the

cutaneous lymphatics give rise to the clinical picture of erysipelas, has not yet been confirmed with certainty.

Undoubtedly cases of erysipelas are by no means rarely encountered in which no wound can be demonstrated, so that the question has been raised whether under some conditions the streptococcus may not gain entrance into the lymphatic vessels through the uninjured skin, perhaps by way of the cutaneous glands. Under such circumstances the designation *true erysipelas* also has been employed, but that of *cryptogenetic erysipelas* would seem to be more appropriate. Under all circumstances the diagnosis of cryptogenetic erysipelas should be made with great reserve. It is to be borne in mind especially that small wounds may already have healed before the symptoms of erysipelas make their appearance. In the majority of cases the wound is an external one, and accordingly *cutaneous erysipelas* develops. Less commonly *erysipelas of a mucous membrane occurs*, as, for instance, the pharyngeal, the laryngeal, the nasal, that of the lacrimal duct, and, in women, that of the uterus. Cases of the last-named variety are included in puerperal fever. Some clinicians even admit the occurrence of *erysipelatous inflammation of the serous membranes and the viscera;* thus, wandering pneumonia is attributed to erysipelatous infection.

Even the minutest wound (needle-prick, leech-bite, vaccination-wound) is sufficient for infection of the skin. The latter will occur the more readily if care be not taken to disinfect all wounds, and to guard them from infection by a protective bandage. Infection may take place by *conveyance through the air.* For this reason it is a common experience that those suffering from wounds are attacked by erysipelas if placed in a room previously occupied by a patient with erysipelas. Streptococci have been demonstrated in the air of such a room, and it is recognized that they may persist for a long time. Occasionally infection takes place through *immediate contact.* It is therefore dangerous to touch patients with erysipelas if one is wounded, particularly on the fingers. Finally, *inanimate objects* and *third persons* may be the means of transmitting the infection. All instruments that have been employed in cases of erysipelas should therefore be thoroughly sterilized and the same statement is applicable to linen, bedding, household articles, and the room. The physician should always visit cases of erysipelas last. In hospitals special physicians and nurses should be provided for cases of erysipelas.

At some seasons, particularly the *spring* and the *autumn*, erysipelas occurs not rarely in *epidemic distribution.* In addition to want of cleanliness in the treatment of wounds the dissemination of the disease is further favored by *overcrowding* and *deficient ventilation of the sick-room.*

Erysipelas is one of those infectious diseases that exhibit a marked tendency to *recur* from time to time in the same patient. This is observed especially in persons suffering from chronic eczema and furuncles of the nose, chronic inflammation of the lacrimal duct, chronic leg-ulcers, or similar processes, which constantly provide a portal for the entrance of the Streptococcus erysipelatosus.

As erysipelas is an *accidental infectious disease*, it is not confined to any special period of life or sex; in the newborn even it may originate in the umbilical wound. Occasionally erysipelas occurs *in conjunction with other infectious diseases*, as, for instance, smallpox, typhoid fever, pharyngeal diphtheria, and syphilis. It sometimes exerts a favorable and curative influence upon the last-named disease. The same statement is applicable, also, to lupus, carcinoma, and leukemia. Therapeutic inoculation with the streptococci of erysipelas has, therefore, been recommended in the treatment of these diseases, although this is not unattended with danger to life.

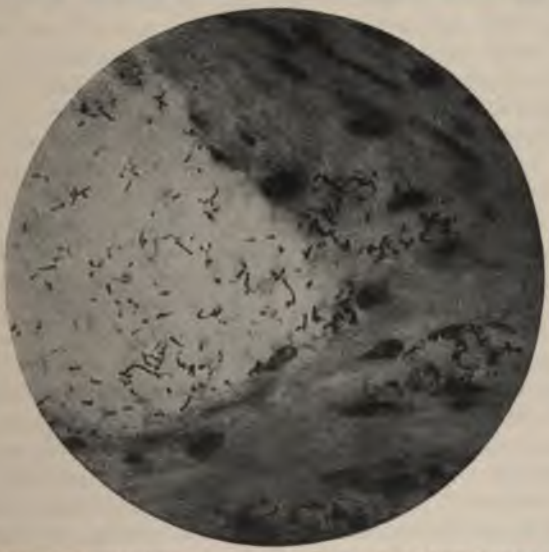


Fig. 58.—Streptococcus erysipelatosus, seen in a section through human skin; × 500 (Fränkel and Pfeiffer).

Symptoms, Anatomic Alterations, and Diagnosis.—The *period of incubation* of erysipelas is often only a few hours. *Premonitory symptoms* (*prodromes*) are wanting, or a severe chill occurs, followed by elevation of temperature to 39° or 40° C. (102.2° or 104° F.), and even higher, often, also, associated with gastric disturbances, particularly vomiting; and these are succeeded in the course of a few hours, at the latest within a day or two, by the characteristic changes in the skin and the mucous membranes. In cases of *cutaneous erysipelas* the attention of the patients is often attracted to the changes in the skin by a sense of **burning** and **prickling**. The skin becomes reddened, feels hot, and is glossy, tense, and tumid. Should the eyelids be attacked by erysipelas they frequently become so greatly swollen that the palpebral fissure is greatly narrowed, or the lids cannot be separated at all. The auricles are thickened and tumid; the lips become swollen into great masses, and the nose also is enlarged and unusually thick. Extensive erysipelas of the face often gives rise to such marked disfigurement that well-known individuals can scarcely be recognized. A tendency for the disease to extend progressively from a small beginning is peculiar to all cases of erysipelas. Facial erysipelas frequently extends to the scalp, where it can be less readily seen than it can be recognized from the swelling and the tenderness of the skin on pressure. The **peripheral lymphatic glands** in the vicinity of the erysipelatous skin are frequently enlarged and become tender on pressure. Inflamed lymphatic vessels also are not rarely visible in the form of reddened strands beneath the skin. Often, but not constantly, the fever and the inflammation of the skin persist for a week, when *critical defervescence* takes place, the swelling of the skin subsiding, the redness and the increased temperature disappearing, and after *desquamation* the skin reacquires its healthy appearance. *Relapses* occur, on the whole, but seldom.

Microscopic examination of the erysipelatous skin discloses hyperemia of the cutaneous vessels, round-cell accumulation about the blood-vessels, and particularly about the lymphatics and the hair-follicles, swelling of the cells of the rête, with round cells between them. Especially distinctive is the demonstration of streptococci in the lymphatics of the cutis, but this is possible only at the periphery, and not in the central portions of the inflamed cutaneous area. *Anomalies in cases of cutaneous erysipelas* relate at times to the degree of general infection and at other times to the nature of the changes in the skin. Occasionally the general infection is so inconsiderable that elevation of temperature does not take place—*afebrile erysipelas*. On the other hand, cases occur in which the elevation of temperature is extremely high, or hyperpyretic. Especially under such conditions, signs of cardiac weakness and exhaustion frequently appear, although such alarming symptoms may develop in conjunction with slighter elevation of temperature.

The designation *vesicular and bullous erysipelas* is applied to cases in which large blebs appear upon the skin, generally, with clear, serous contents; at times, however, also containing pus—*pustular erysipelas*. Left to themselves, either the blisters rupture or their contents undergo desiccation into thin crusts, which fall off in the course of a few days without leaving cicatrices. Further, it will be observed that small, often imperfectly developed cutaneous vesicles are demonstrable in most cases of cutaneous erysipelas. The variety of erysipelas designated *gangrenous* is of serious prognostic significance. In consequence of excessive swelling and tension of the skin so marked a degree of compression of the blood-vessels results that the skin undergoes necrosis and becomes black and gangrenous. In consequence destruction of the skin occasionally results with remarkable rapidity, and, besides, such a marked degree of exhaustion becomes manifest as to cause death from collapse.

Although cutaneous erysipelas always exhibits a marked tendency to spread, the designation *wandering* or *migratory erysipelas* is employed only when this tendency attains an unusually marked degree. In such an event the inflammation may extend gradually from a circumscribed area of the skin over the entire body, areas of skin that have recovered may be again attacked, and the disease may be protracted for many weeks. Under such circumstances death may result from exhaustion.

Among the *complications of cutaneous erysipelas* inflammatory processes in any viscus should be mentioned. *Purulent meningitis* is to be considered as of especial importance, occurring sometimes as a complication of erysipelas of the face and of the scalp. Erysipelas of the face and of the neck is occasionally attended with the sudden development of symptoms of *acute edema of the glottis*, which at times may terminate life speedily. *Inflammation of the middle ear* and *acute nephritis* also are among the more common complications. Among the *sequelæ*, *loss of hair* should not be forgotten, occurring usually after erysipelas of the scalp. Generally, however, the hair grows again in abundance, as the condition is dependent upon nutritional disturbances of a transitory character. Occasionally cutaneous erysipelas is succeeded by obstinate *neuralgia*. If erysipelas has occurred repeatedly in the same situation, *elephantiasic thickening of the skin* readily develops.

Little of a definite nature is as yet known with regard to *erysipelas of the mucous membranes*. Doubtless many cases of *phlegmonous angina* and *acute edema of the glottis* are but expressions of erysipelas of the mucous membranes; but the diagnosis can be made positively only if the symptoms mentioned occur as a complication of, or are complicated by, cutaneous erysipelas.

Prognosis.—The prognosis of erysipelas is not unfavorable, and a fatal result is rather exceptional. When this occurs it is generally due to the severity of the general infection or to grave complications.

Treatment.—No specific treatment for cutaneous erysipelas is known. Injections of streptococcus-serum have as yet not yielded brilliant results. In most cases expectant treatment will suffice, Oily inunctions are agreeable to most patients, as they leave behind a sense of coolness. It is our custom to prescribe generally a carbolated ointment:

℞ Carbolic acid, 5.0 (75 grains);
Wool-fat,
Lard, each, 25.0 (⅘ ounce).—M.
To be used by inunction twice daily.

The **diet** should be liquid. When thirst is marked phosphoric acid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—every two hours) may be given.[1]

Symptomatic treatment should be instituted if certain symptoms acquire threatening proportions. Persistent elevation of temperature above 40° C. (104° F.) will indicate, especially in drunkards, in the aged, in patients suffering from heart-disease, and in pregnant women, the administration of *antipyretics*, among which first place should be given to phenacetin (1.0—15 grains). In order to restrain the wandering tendency of erysipelas the borders of the cutaneous inflammation have been painted with tincture of iodin or silver nitrate; or subcutaneous injections of carbolic acid (2 per cent.) have been made; or strips of adhesive plaster have been applied; but the result is always uncertain.

Great importance is to be attached to the *prophylaxis*. All wounds, even the smallest, should be cleansed with carbolic acid (from 3 to 5 per cent.), or with mercuric chlorid (0.1 per cent.), and guarded against subsequent infection by the application of a protective bandage. Chronic inflammatory processes in the nose, the ear, and the lacrimal duct, as well as leg-ulcers, should be relieved. Cases of erysipelas should always be isolated in hospitals, and be provided with special attendants, physicians, and instruments. After the discharge of the patient the sick-room, together with its contents, should be thoroughly disinfected. Occasionally erysipelas develops after vaccination. Under such circumstances the practise of vaccination should be suspended for a time. In the presence of *erysipelas of the mucous membranes ice* should be employed. Tonsillar abscesses should be incised with the knife, and if edema of the glottis threaten, intubation or tracheotomy should be practised.

HERPES ZOSTER.

Etiology.—Herpes zoster is attended with the presence of *groups of vesicles* that appear in the *area of distribution of certain*

---

[1] In sthenic cases hypodermic injection of pilocarpin (gr. $\frac{1}{6}$) once daily, or oftener, has yielded admirable results; fluid extract of pilocarpus (from 15 to 30 minims thrice daily) also may be given. Applications of ichthyol (50 per cent. with wool-fat) are likewise most effective. Good results are reported also from the subcutaneous injection of mercuric cyanid (gr. $\frac{1}{6}$) at the periphery of the inflammatory area. In asthenic cases it is well to administer a combination of iron and quinin.—A. A. E.

nerves. These lesions occur upon the skin and the mucous membranes, so that a distinction has to be made between an *exanthematic* and an *enanthematic* variety of herpes zoster. The actual causes for herpes are to be found in nervous disturbances, frequently in neurotic processes, and, accordingly, herpes has often been considered a result of trophic disturbances. Nervous disturbances and herpes zoster may, in the first place, occur as a result of **mechanical irritation,** and herpes zoster has therefore been observed in the sequence of lesions of a peripheral nerve (contusion, extraction of a tooth). The employment of excessively strong galvanic currents also has brought about an eruption of herpes zoster. Occasionally the condition is dependent upon **neuritis resulting by extension from adjacent disease.** In this way herpes zoster may be secondary to tuberculosis and carcinoma of the vertebral column, and under these conditions inflammation of the intervertebral ganglia has in a number of instances been demonstrated.

The *toxic varieties* of herpes zoster include the cases in which the disorder has developed after the employment of arsenic, or after carbon-monoxid poisoning. An *autotoxic variety* of herpes zoster has been observed in the course of uremia. It occurs also in cases of diabetes mellitus. *Infectious herpes zoster* finally remains to be mentioned, and this may be either primary or secondary. The latter variety develops in the course of a number of infectious diseases (pneumonia, erysipelas, rarely typhoid fever), while the primary variety occurs as an independent infectious disease, occasionally in epidemic distribution, and particularly in the autumn and the spring. One attack of herpes zoster almost always protects against subsequent attacks.

Symptoms, Anatomic Alterations, and Diagnosis.—*Herpes zoster of the skin* is often unattended with fever, although **neuralgia** in the course of the nerve in whose distribution the vesicles appear is often an antecedent condition. In some cases, it is true, the neuralgia appears only after the development, or even after the subsidence, of the vesicles. The patient may discover the vesicles accidentally, or his attention may be attracted to the changes in the skin by a sense of **prickling** or **burning.** The **exanthem** consists of vesicles varying in size from that of a pinhead to that of a lentil, which are surrounded by a red areola and often are umbilicated, and are arranged closely together in groups of from ten to thirty or more. It is distinctive for these groups of vesicles to follow the distribution of certain nerves. It is true it has recently been contended that the vesicles do not follow the distribution of the nerves, but that of the cutaneous vessels. Herpes zoster is almost always a *unilateral* disorder, and only exceptionally is it bilateral. The skin between the groups of vesicles appears unaltered, but in some instances it is found to be anesthetic or hyperesthetic. Often individual groups are but imperfectly developed, and consist rather of papules than of vesicles. In from three to seven days the serous contents of the vesicles dries up into thin yellowish or brownish crusts, which soon fall off without leaving cicatrices. At first there remain upon the skin round whitish spots that are generally surrounded by a zone of brownish pigment, but after the lapse of a few weeks the skin resumes its normal appearance. The neuralgia occasionally persists for a long time beyond the exanthem, and at times even for years.

Intercostal herpes zoster is the variety most frequently encountered, developing generally in the distribution not of a single intercostal nerve, but of two or three contiguous nerves, and involving with especial frequency the third, fourth, and fifth intercostal nerves. At the same time it will readily be observed that the groups of vesicles often extend for a short distance beyond the median line, both posteriorly as well as anteriorly, a feature that is common also to herpes in the course of other nerves. On the head herpes zoster occurs most frequently in the course of some of the branches of the trigeminal nerve, but in connection with disease of the trigeminal trunk throughout the distribution of the entire nerve. *Ophthalmic herpes zoster* is not rarely of grave significance, as it may within a short time be complicated by softening, inflammation, and destruction of the eyeball. These changes have been considered as the result of trophic disturbances, a view that, naturally, has not been unopposed.

Among the *anomalies of herpes zoster hemorrhagic herpes zoster* and *gangrenous herpes zoster* may be mentioned. The contents of the vesicles in cases of hemorrhagic herpes zoster are not serous, but bloody. This is, on the whole, a matter of small importance, particularly if the contents of but a few groups of vesicles are hemorrhagic. Gangrenous herpes zoster is a graver condition. The vesicles and the adjacent skin acquire a blackish, gangrenous aspect, and destruction of the skin results, occasionally extending deeply into adjacent tissues, healing slowly, and leaving permanent cicatrices. In addition, this variety of herpes is not rarely attended with intense pain and such alarming exhaustion that death from collapse may be threatened. Fortunately, gangrenous herpes zoster occurs but rarely.

Complications are rare, apart from the *neuralgia* mentioned. Occasionally paralysis, central or peripheral, has been observed. Paralysis of individual nerves has corresponded at times to the distribution of the herpes, at other times to that of adjacent nerves. In one case I observed facial palsy to precede herpes of the trigeminal nerve. The same conditions also are the most common *sequelæ*.

Among the various forms of *herpes of the mucous membranes,* *herpetic angina* may first be mentioned. This sets in not rarely with chill and fever, and is attended with severe pain in the pharynx, particularly in swallowing. The characteristic lesion consists in groups of vesicles, of unilateral distribution, and occupying one-half of the uvula or one of the arches of the palate. The diagnosis can be made only with the aid of the laryngoscope if the herpetic vesicles are situated upon the posterior aspect of the uvula. Herpetic angina occurs occasionally in epidemic distribution, and at times is followed by paralysis of the palatal muscles, rarely also by perforation of the palate. In some cases it occurs as a complication of facial herpes zoster, or it may be associated with *herpetic stomatitis and glossitis*. *Herpetic laryngitis* can be detected only with the aid of the laryngoscope. The groups of vesicles occupy especially one-half of the epiglottis and the mucous membrane of the true vocal bands.

Progenital herpes may yet be mentioned, occurring in men upon the inner layer of the prepuce, occasionally also upon the glans and the dorsum of the penis. It occurs especially in those in whom the prepuce is tight, so that sebaceous matter readily collects beneath it and undergoes decomposition, also in those in whom the glans and the prepuce are but rarely, if at all, cleansed, or who engage in sexual intercourse with women suffering from irritating discharges from the genital organs. The attention of the patient is attracted to the disorder by a sense of burning, prickling, and itching in the prepuce, to which redness and swelling may be superadded. Progenital herpes comes under the observation of the physician less commonly in the form of vesicles than in that of shallow ulcers resulting from rupture of the vesicles and healing within a few days, without the formation of cicatrices. The patient generally consults the physician because he thinks the superficial lesion of the skin to be a chancre. The differentiation is easy because herpetic ulcers may occur even without previous sexual intercourse, and they heal within a few days in the sequence of expectant treatment. Progenital herpes occurs repeatedly in some men, as this variety of herpes zoster has a tendency to return. Corresponding lesions occur, also, in women on the inner aspect of the labia minora and on the prepuce of the clitoris.

Prognosis.—Herpes zoster is a benign disease, which terminates fatally with extreme rarity. Nevertheless death has been observed to occur amid profound coma, particularly in cases of trigeminal herpes zoster.

Treatment.—No *specific treatment* is known, and in view of the benign character of the disorder is also not necessary. In a case of herpes zoster of the skin it will suffice to anoint the vesicles with olive-oil, and then to cover them with absorbent cotton, in order to protect them against rubbing. The presence of severe neuralgia may require the employment of quinin, arsenic, subcutaneous injections of morphin, or the use of the galvanic current. In cases of trigeminal herpes zoster, with ophthalmia, the eye should be protected by a bandage, inasmuch as, according to the view of some clinicians, the ocular condition is dependent not upon trophic disturbances, but upon anesthesia of the cornea, so that foreign bodies, particularly bacteria from the air, readily find lodgment upon it and enter upon their work of destruction. In cases of *herpetic angina*, *stomatitis*, and *glossitis* mouth-washes and gargles of potassium chlorate (5.0 : 200) or solution of aluminum acetate (1.0 : 200) may be prescribed. *Progenital herpes* should be treated in the same way as herpes of the skin. In order to prevent its recurrence the prepuce should, if necessary, be stretched and washed regularly.

MILIARY FEVER.

Miliary fever is known also as the *English sweating sickness*, because the disorder was first observed in England. It is a rare affection, whose independent character is disputed by some physicians. The *period of incubation* is placed at two or three days. A *prodromal period* of equal length then often follows, being attended especially with great malaise and languor. The first appreciable manifestations consist in profuse *sweating*, which generally sets in during the night, and persists in association with a sense of fear, palpitation of the heart, dyspnea, and often, also, with cramps in the calves. In the course of a few days the skin becomes covered with innumerable *miliary vesicles*, which undergo desiccation at the end of the first week of the disease, and this is followed by slight desquamation. The *bodily temperature*, which at first is elevated, then declines to the normal level. The dangers of the disease consist in *exhaustion* or in serious *complications* (pneumonia, diphtheria). No specific remedy is known. The patients should not be kept too warm, should be given wine diluted with water, and for the suppression of the sweating atropin sulphate (0.01 : 10—$\frac{1}{6}$ grain : 2$\frac{1}{2}$ fluidrams; from one-half to one syringeful—from 8 to 15 minims—once or twice daily) should be injected subcutaneously.

VARICELLA (CHICKEN-POX).

Etiology.—Chicken-pox is so pronounced an infectious disease of childhood that practically it is well not to diagnose the disease at all in adults, but to consider suspected chicken-pox in the latter as mild small-pox (variola). The *infective agent* of chicken-pox is unknown. Nevertheless it is present, as successful experimental inoculation of healthy persons has shown, in the contents of the varicellous vesicles.

Some physicians believe that chicken-pox is merely a mild variety of small-pox, but opposed to this view is the fact that vaccination confers protection only against small-pox, and not against chicken-pox; that small-pox may occur after an attack of chicken-pox, and *vice versâ;* and that vaccination will be successful also in children who have recovered from chicken-pox.

The disease is most frequently acquired by children in *schools* and on *playgrounds* through immediate *association* with the sick; or, at a short distance, through the *intermediation of the air.*

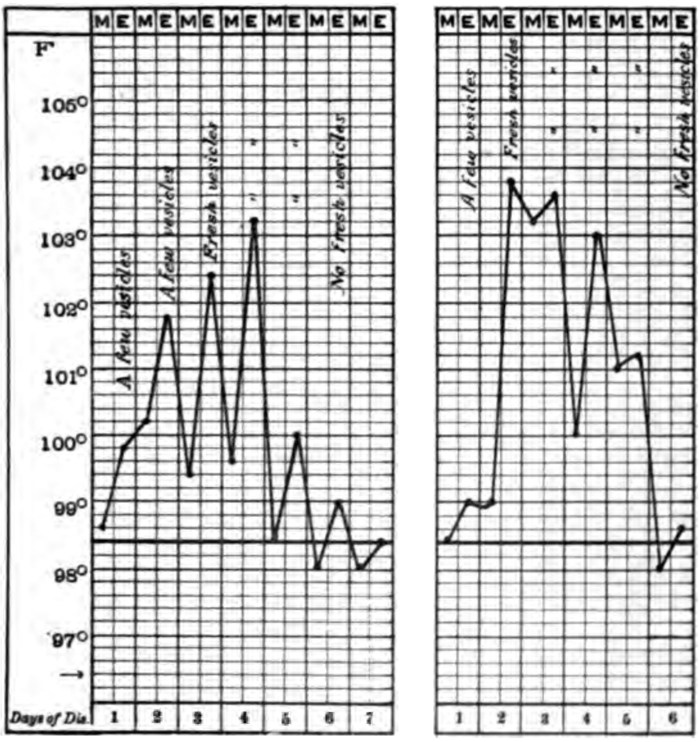


FIG. 59.—Temperature charts from cases of varicella (Ashby and Wright).

Third persons and *inanimate objects* also are capable of spreading the disease. Chicken-pox may occur *sporadically*, *epidemically*, or *endemically*. Endemics of chicken-pox are, for instance, not rarely observed among the pupils of one school, in boarding-houses, or orphan-asylums. Epidemics of measles, whooping-cough, or scarlet fever often occur in conjunction with those of chicken-pox, or epidemics of the diseases named may replace and follow one another. Recovery from an attack of chicken-pox generally confers *acquired immunity* from future attacks, so that a *repetition* of the disease occurs rarely, and is noteworthy.

Symptoms, Anatomic Alterations, and Diagnosis.—The *period of incubation of chicken-pox* is generally fourteen days. *Prodromes* are wanting, or the children become pale, peevish, without appetite, and exhibit slight febrile movement. The characteristic **exanthem** usually appears earliest on the face, but gradually invades also the trunk and the extremities. It is peculiar that the eruption of the vesicles of chicken-pox may extend over more than two or three weeks, so that the first vesicles may already be healed while new changes in the skin are taking place in other portions of the body. Red, roundish, flat papules, as large as a pea, and larger, first appear upon the skin, and the overlying epidermis becomes raised in the form of vesicles, which generally exhibit a distinct depression (umbilicus) at the center. The contents of the vesicles are at first transparent and serous, but subsequently they become opaque, milky, even slightly purulent, as an increasing number of round cells are admixed. In the course of a few days (from three to five) the vesicles undergo desiccation into grayish or brownish crusts, which fall off in from three to seven days, without leaving cicatrices. Cutaneous cicatrices occur exceptionally only if isolated varicellous vesicles are not confined to the thickness of the epidermis, but extend into the cutis; or if the patient has scratched some vesicles and has thereby also injured the cutis. The number of vesicles is susceptible of great variation.

In *microscopic structure* the vesicles of chicken-pox resemble those of small-pox. The vesicles are situated between the horny layer and the mucous layer of the epidermis, and exhibit an alveolar arrangement, in the interstices of which the fluid contents are present. The trabeculæ of the vesicles consist of compressed and degenerated epithelial cells.

In a large number of cases an **enanthem** is present in addition to the exanthem. A number of white vesicular elevations, surrounded by a reddened areola, are found with especial frequency upon the mucous membrane of the tongue, the palate, and the pharynx, although they may occur also upon the laryngeal and the conjunctival mucous membrane, and upon the prepuce and the labia.

The *general condition* often is scarcely affected. Some patients complain of a sense of **burning and prickling in the skin.** The **peripheral lymphatic glands** also are often enlarged and tender on pressure. **Fever** occasionally is wholly wanting; at any rate, it is the rule that elevation of temperature when present is not high, and persists for but a few days. The *course of the disease* is occasionally protracted, extending over more than four weeks.

Among the *anomalies of the exanthem* ***hemorrhagic*** and ***ventose*** *varicella* may be mentioned as indifferent variations. In the first some of the vesicles contain blood; in the latter, air. *Gangrenous varicella* is of serious significance, occurring especially in poorly nourished, tuberculous (scrofulous) children, and not rarely terminating fatally from exhaustion. *Complications* (erysipelas, acute nephritis, polyarthritis, neuritis, etc.), are rare. *Sequelæ* also do not occur generally. In isolated instances paralysis has developed in consequence of neuritis.

Prognosis.—Varicella is a benign disease, which is but rarely attended with danger to life.

Treatment.—The treatment should be confined to tepid **baths** (35° C., 28° R., 95° F., morning and night) and a light **diet**, which should be **liquid** in the presence of febrile movement. It is wise, even in the absence of fever, to keep the children in bed. In order to prevent dissemination of the disease it is well to *isolate* the patient, and, when recovery has taken place, to permit him to associate with others only after the bed-linen, the body-linen, the room, and the furniture have been sterilized (disinfected) in the manner described on p. 316.

VARIOLA (SMALL-POX).

Etiology.—Small-pox justly was greatly feared until the beginning of the nineteenth century, because hundreds of thousands annually were made its victims, or at least were deformed or distorted by its effects. Since the introduction of *vaccination* the disease has almost lost its terrors for those who have submitted to this protective measure, as vaccinated individuals remain almost permanently exempt from small-pox, or exceptionally are at most attacked by a mild and harmless form of the disease if they have failed to be revaccinated from time to time. The certain protection of vaccination, thus, does not extend over more than five, and at most more than ten, years. In countries in which vaccination is prescribed universally by law small-pox occurs but rarely, and then it is generally imported from such country in which protective inoculation is practised indifferently if at all.

The *virus of small-pox* is as yet unknown. It is not surprising that pyogenic cocci (staphylococci, streptococci) are found in the purulent contents of the pustules. Inoculation-experiments on human beings have shown that the infective material resides in the purulent contents of the pustules and in the crusts resulting from desiccated pustules. As a matter of course, the secretions from mucous membranes also acquire infective properties when contaminated by the contents of the pustules. *Infection* takes place in the same manner as with most infectious exanthemata—namely, through immediate *contact*, and the intermediation of the *air*, of a *third person*, and of *inanimate objects*. Through the last small-pox may be carried for great distances. Thus, small-pox appeared suddenly among the operatives in a feather-bed factory in Zurich. Careful investigation disclosed that the feathers had been derived from Russia and Poland, and from places in which small-pox prevailed. The same relation has been observed among rag-sorters.

Most individuals possess, as for measles, a *congenital predisposition* also for small-pox, and the danger of the disease is thereby increased. This natural predisposition can be neutralized only by vaccination. Recovery from an attack of small-pox confers *acquired immunity* from subsequent attack comparable to that conferred by vaccination. A *second attack of small-pox* is exceedingly rare. If all persons were revaccinated every five years, it would probably be possible to cause the disappearance of small-pox from the earth. For some incomprehensible reason many do not submit to vaccination; and even some countries, which occupy otherwise a front rank in humane provision, such as the canton of Zurich, have inexplicably abolished compulsory vaccination, so that a source for small-pox is thus constantly maintained. In countries in which vaccination is made obligatory by law epidemics of small-pox scarcely occur. Should cases of small-pox be introduced from without, they may occasionally give rise to infection of individuals about them who have not been revaccinated for a long time; but susceptible persons capable of causing epidemic distribution are wanting. The conditions are different in countries in which vaccination is not compulsory. In 1884 small-pox was introduced into the canton of Zurich from Marseilles, and an epidemic resulted which persisted into the year 1886, and involved 719 persons. *Epidemics of small-pox* recur at certain intervals in countries in which vaccination is not compulsory, as, for instance, from every two to every four years. Occasionally individuals are attacked simultaneously with *small-pox and some other infectious disease*, as, for instance, typhoid fever, syphilis, etc.

Symptoms.—The *period of incubation of small-pox* is, on the average, ten days. The prodromes begin, as a rule, with a chill, which is followed by elevation of temperature to 39° or 40° C. (102.2° or 104° F.) and even more. Frequently symptoms of gastric derangement appear, particularly repeated vomiting. Severe **pain in the lumbar region**, however, is especially marked, but with regard to whose seat (kidneys or meninges of the lumbar cord) and cause (hyperemia) there is no certainty. In some cases fugacious **prodromal exanthems** appear, generally erythemata in the hypogastric region and upon the external aspect of the legs. The prodromal stage generally continues for three days, and in this connection it should be noted that the bodily temperature declines from day to day, so that the patients often believe themselves convalescent from the disease, and attempt to resume their work. Also with the beginning of the *stage of eruption* elevation of the bodily temperature frequently does not at once take place. The stage of eruption, which lasts about fourteen days, has been divided into several substages in accordance with the development of the cutaneous eruption, and these have been designated macular, papular, vesicular, pustular, and encrusted.

In the *macular stage* circumscribed, rounded red spots appear upon the skin, which become pale upon pressure with the finger, and therefore are dependent upon circumscribed hyperemia of the skin and are roseolæ. These spots may attain the size of a pea and even beyond. The patient completely resembles one suffering from measles, and the more so as the variolous spots also appear earliest on the face, and in particular abundance upon the forehead and the cheeks, and then, within twenty-four hours, extend to the trunk and the extremities. It is therefore not possible during the first twenty-four hours to distinguish from the exanthem a case of measles from one of small-pox. In favor of small-pox are the prevalence of an epidemic of this disease and the occurrence of prodromal pains in the loins. In the course of a day the conditions naturally are more clearly defined, because in a case of small-pox acuminated papules appear upon the individual roseolæ, and the macular stage is followed by the *papular stage*. This stage also persists for but a day.

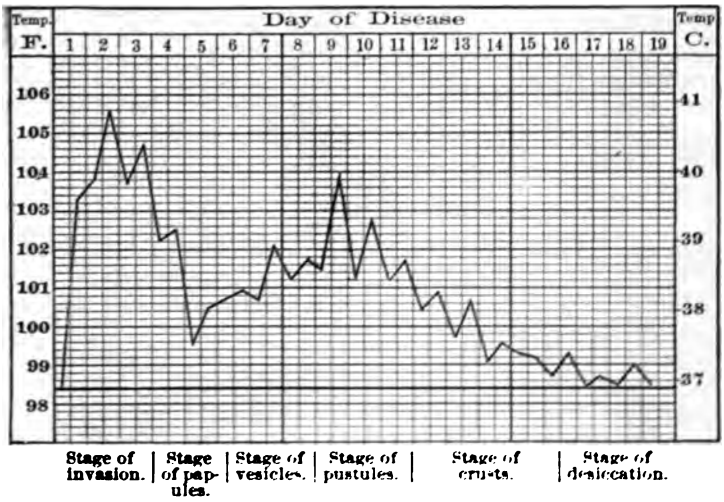


FIG. 60.—Temperature-curve of small-pox (Salinger and Kalteyer).

With the elevation of the epidermis upon the individual papules in the form of vesicles the *vesicular stage* develops, and this in the course of about three days passes over into the *pustular stage*. In the latter the fully developed variolous pustule is present. This lesion attains on an average the size of a pea, and occurs as a milky-cloudy or markedly purulent vesicle, which exhibits a distinct depression at the center, known as the *umbilicus* or *indentation*, while it is surrounded by a red areola (halo) several millimeters wide. On the third day of the pustular stage the exanthem has reached the height of its development. There then follows the stage of desiccation, or the *encrusted stage*, with which the entire stage of eruption terminates. The purulent contents of the individual pustules dry up and form greenish or brownish crusts. Some pustules also rupture, and the discharging contents form crusts of the character described. The crusts remain upon the skin for from five to seven days on the average, so that the total duration of the stage of eruption is about fourteen days.

The disease terminates with the *stage of decrustation and desquamation.* The desiccated crusts gradually fall off and desquamation of the epidermis takes place, persisting for about fourteen days. When the disease pursues a regular course a patient suffering from small-pox will have recovered at the end of the sixth week following infection. After the crusts have fallen off the skin at first is mottled with red spots that have remained at the site of the pustules, and which disappear only in the course of weeks, because their color is due in part to extravasated red blood-corpuscles. It occurs frequently, however, that the pustules have invaded the cutis, and under such circumstances they are followed by deep excavated *cicatrices*, which persist throughout life and, if present in considerable number, cause permanent disfigurement.

As with all infectious exanthemata, so also in the case of small-pox, lesions of the mucous membranes are encountered, a *variolous enanthem* in addition to the exanthem. These occur not rarely as early as the prodromal period, and at times assume the characters of diffuse *catarrhal inflammation*, and at other times of *variolous pustules*. Many patients complain of a sense of burning, of the presence of a foreign body in the eye, and of lacrimation, and under such circumstances the conjunctival mucous membrane is found greatly reddened and swollen, occasionally even chemosis of the conjunctiva. Corresponding alterations and manifestations occur also upon the nasal, the pharyngeal, the laryngeal, and the tracheobronchial mucous membranes, and give rise respectively to sneezing, a sense of burning in the nose, a nasal discharge, pain in swallowing, hoarseness, cough, and expectoration.

Variolous pustules appear most frequently upon the mucous membrane of the hard palate and the pharynx, and form whitish, generally wrinkled vesicles with a red areola. They are not rarely observed also upon the tongue. Under such circumstances the tongue often becomes increased in size and its surface heavily coated, while its borders exhibit the impressions of the teeth—*variolous glossitis.* There exists also salivation, and a disagreeable odor emanates from the mouth. Variolous pustules in the pharynx cause intense pain in swallowing, and upon the tongue pain in speaking and eating.

Pustules upon the laryngeal mucous membrane give rise to hoarseness and pain in the larynx. Occasionally they cause acute inflammatory swelling of the aryepiglottic folds and false vocal bands—so-called acute edema of the glottis—which may induce death from suffocation within the shortest possible time. A further danger from endolaryngeal variolous pustules resides in their extension deeply, with the development of laryngeal perichondritis and its sequels. At times adhesions form between apposed surfaces of mucous membrane, with the development of membranous and septum-like stenosis of the larynx, the correction of which is generally attended with great difficulty.

Variolous pustules upon the *ocular conjunctiva* may be followed by severe catarrhal, and not rarely also by suppurative, inflammation. The conditions are also serious with regard to the eye if pustules are situated close to the periphery of the cornea or even extend to the cornea, as suppurative inflammation and destruction of the entire eyeball (panophthalmitis) may readily result. Under such circumstances blindness will occur.

Epidemics of small-pox especially were justly feared in previous centuries on account of the frequent occurrence of bilateral panophthalmitis and bilateral blindness. Pains and roaring in the ears and impairment of hearing are often dependent upon inflammation of the mucous membrane of the Eustachian tube and the tympanic cavity.

Pustules upon the mucous membrane of the esophagus and the stomach may remain latent during life. In some cases symptoms of esophageal stenosis have been observed after cicatrization had taken place. *Pustules upon the urethral mucous membrane* are attended with burning and pain during and after the act of micturition. *Pustules upon the vaginal mucous membrane*, the *cervix uteri*, and upon the *mucous membrane of the rectum* can be recognized with the aid of a speculum.

The **bodily temperature** pursues a markedly peculiar course in cases of small-pox. As has already been mentioned, the prodromal elevation of temperature at the beginning of the stage of eruption is frequently but slight. Only in the period of suppuration, thus four days after the appearance of the exanthem, does the temperature again rise to a higher level, to fall again slowly to the normal, however, in the course of one or two days. The stage of desquamation is unattended with fever.

Relapses of small-pox occur but rarely. Among the *anomalies of small-pox* may be mentioned *varioloid* and *confluent variola*. *Varioloid* represents a mild and benign form of small-pox, which occurs especially in vaccinated individuals when attacked by small-pox. Often but a few pustules appear upon the entire body, and the general condition and the bodily temperature are often not at all altered. *Confluent variola,* on the other hand, represents a severe degree of disease. The pustules are so close together that they coalesce in many places. Should desiccation of the pustules take place, a coherent crust forms especially upon the face and the hands, which covers the face like a mask, and gives rise to hideous disfigurement, while the hands and the fingers are slightly curved and immobile, because all movement is attended with intense pain. If the crusts are punctured, an abundance of greenish, creamy pus escapes, which frequently yields a disagreeable odor. Some patients contaminate the atmosphere of a room to an intolerable degree within a short time. In conformity with the marked development of the exanthem, the general condition also is profoundly affected. Particularly at the period of pustulation (suppuration) the bodily temperature often rises considerably, and also persists elevated for a longer time than usual, and is attended with such serious exhaustion that death from collapse is not a rare occurrence. This severe variety of small-pox occurs almost exclusively in the unvaccinated.

Hemorrhagic variola is a grave form of small-pox. It is characterized by the abundant occurrence of hemorrhages into the skin, varying in size from punctate petechiæ to extensive extravasations (ecchymoses, ecchymomata). In addition, internal hemorrhages often occur, as, for instance, epistaxis, hematemesis, hemoptysis, hematuria, enterorrhagia. The patients fail rapidly, become comatose and delirious, and often succumb speedily from excessive exhaustion.

Abortive variola is one of the mildest varieties of small-pox. This occasionally terminates with the prodromal manifestations, or the exanthem develops only to the macular or the papular stage.

Complications occur with extreme frequency in the more severe cases of small-pox, and reference has already been made to serious *involvement of the eye, the ear, and the larynx.* Inflammatory lesions may, however, develop in any of the internal organs, although no definite inflammatory process can be said to occur with especial frequency. It should be mentioned, further, that occasionally mental disease develops during the presence of the exanthem or after its subsidence. Neuritic, myelitic, and encephalitic lesions also are known to occur as complications, and still more frequently as sequelæ.

Among the *sequelæ* the *cicatrices* are the most frequent. Occasionally these are followed by disfiguring *keloids.* It has already been mentioned that *disorders of the eyes and the ears* persist. In general, complications of small-pox not at all rarely give rise to permanent sequelæ.

Diagnosis.—The recognition of small-pox is not difficult.

In the macular stage, it is true, **measles** cannot be distinguished from small-pox, although the conditions become clear in the course of twenty-four hours if acuminated papules have appeared upon the questionable macules of measles, and these soon are converted into vesicles and pustules. In doubtful cases sacral pains should be inquired for during the prodromal period, as these are distinctive of small-pox, and, also, whether the patients have been associated with others suffering from measles or from small-pox, and whether an epidemic of measles or of small-pox is prevalent in the place or in its vicinity.

Occasionally pustules appear upon the skin is cases of **nodose erythema**, and these may resemble the lesions of small-pox, so that it is necessary to hold the diagnosis in reserve for a few days until the appearance of distinctive erythematous nodes, or their failure to appear, determines the diagnosis. In the presence of **syphilids** resembling the eruption of small-pox examination of the genitalia for syphilitic lesions should not be omitted. In cases of **contagious impetigo** the general condition is but little affected, and the exanthem is frequently confined to the face; while in cases of **scabies**, which, if attended with fever, may be suggestive of small-pox, the burrows of the acarus can be demonstrated on certain portions of the skin, and in them ova and parasites.

Pustules may be produced artificially upon the skin by an **ointment of antimony and potassium tartrate**, and these may wholly simulate the appearance of variolous pustules, whence the ointment named has been designated also *variolous ointment*. The history and the absence of fever are decisive in the differential diagnosis. In the differential diagnosis between small-pox and **chicken-pox** the age of the patient should first receive consideration, as chicken-pox occurs almost exclusively in children. Besides, the general symptoms of chicken-pox are exceedingly mild.

Prognosis.—The prognosis of small-pox depends upon the severity of the exanthem and the general condition, and upon such complications as may be present: or, what amounts to the same thing, whether the disease occurs in vaccinated or in unvaccinated individuals. Pregnancy constitutes a serious complication, as pregnant women are attacked with especial frequency by confluent variola, not rarely by hemorrhagic variola, and abortion and puerperal septicemia with a fatal termination often occur. The child occasionally is born with a well-developed variolous eruption, and soon dies, or it is seized with small-pox shortly after birth. Should pregnancy not be interrupted, the child may undergo variolation in the uterus, which can be recognized from the circumstance that subsequently vaccination cannot be accomplished, although the infants nevertheless remain exempt from small-pox.

Anatomic Alterations.—The *variolous pustule* develops between the horny and the mucous layer of the epidermis, and exhibits an alveolar structure, the individual alveoli containing pus-corpuscles. The mode of development of the umbilication has not yet been satisfactorily explained. Beneath the pustule the blood-vessels of the cutis are dilated, in places in the form of ampullar enlargements, and surrounded by accumulations of round cells. The **internal organs** exhibit nothing that is characteristic of small-pox, and similar alterations are observed as attending other acute infectious diseases: dark-red, ham-colored, and dry muscular tissue, with granular turbidity, fatty and waxy degeneration of some of the muscle-fibers, flabby heart with the microscopic changes in the fibers of the myocardium just mentioned, enlargement of the spleen, and frequently also of the liver, granular turbidity and fatty degeneration of the liver-cells, the epithelial cells of the convoluted uriniferous tubules, the glandular cells of the gastric and intestinal mucous membrane, and of the pancreas. Necrotic areas and collections of round cells are occasionally encountered in the liver and the kidneys.

Treatment.—No specific remedy for small-pox is known, as *blood-serum therapy* has not as yet proved practicable, and the greatest importance therefore is to be attached to a trustworthy *prophylactic procedure*—namely, *vaccination*—whose beneficent results can be doubted only by persons with extremely narrow views, or incompetent to form an opinion of medical experience and facts. In order to secure immunity from infection with small-pox with certainty vaccination should be repeated every five years, and not later than every ten years, as the protective influence does not persist in some persons for a longer period. A patient suffering from small-pox should be treated in a **special hospital** standing alone, and as remotely as possible from other occupied dwellings. The family of the patient should be quarantined from general association with others for fourteen days, as some of its members may be already infected, and possibly not present symptoms of the disease before the lapse of two weeks, although during this time they may transmit the disease to all persons with whom they come in contact. Dwelling-rooms and furniture should be disinfected in the usual manner. If a small-pox patient die, the body should be wrapped in sheets saturated with a 5 per cent. solution of carbolic acid, and be placed in an hermetically sealed coffin. Exposure of the body, which remains infective, is not permissible. The burial should be private, and without unnecessary attendants.

The small-pox patient should receive only **a liquid diet** so long as fever is present. After each meal the **mouth** should be thoroughly rinsed with **potassium chlorate** (5.0 : 200) or **solution of aluminum acetate** (1 : 200); and, if pustules have developed in the mouth, the pharynx, or in the larynx, bits of ice should be swallowed, and an ice-bag (ice-collar) should be placed about the neck. Internal and external medicaments are without influence upon the development of small-pox. It has been maintained that red light, therefore exclusion of the chemic rays of sunlight, prevents the development of the pustules, and, accordingly, it would be necessary for the windows in the sick-room to be constructed of red glass, although I have had no personal experience with this method. Baths of mercuric chlorid also are said to prevent the development of the pustules. The employment morning and night of tepid **baths** (35° C.—28° R.—95° F.) is warmly to be recommended. In cases of confluent variola it is occasionally well to incise the crusts in places with a knife in order to permit the escape of the retained pus. Inunctions with fats, or hot cataplasms, also at times become necessary in order to soften the crusts and to favor their more ready exfoliation. Grave symptoms should be relieved in the usual manner. The small-pox patient should not be permitted to leave the hospital until desquamation of the skin has been completed, and naturally only with disinfected clothing and linen.

II. INFECTIOUS DISEASES ATTENDED WITH LOCAL ALTERATIONS IN THE LOCOMOTOR APPARATUS (JOINTS AND MUSCLES).

ACUTE ARTICULAR RHEUMATISM.

Etiology.—Exposure to cold (rheuma) was formerly considered the cause of acute articular rheumatism. Recently, however, the disease has been properly included among the *infectious diseases*, and the time has therefore arrived when the old inappropriate designation may be dropped, and possibly be replaced by that of acute primary polyarthritis.

In accordance with past experience **various bacteria** appear capable of inducing polyarthritis, as *pyogenic streptococci* and *staphylococci* have been found in the articular exudate. In some cases, it is true, the exudate was sterile, but in the first place it may have become so with the lapse of time, and, besides, the exudate may possibly contain no bacteria, although these may be present in the tissues of the capsule of the joint.

Acute polyarthritis is a rare disease in childhood, although it has occasionally been observed even in the newborn whose mothers have suffered from acute polyarthritis. It occurs most frequently between the *fifteenth and the fortieth year of life*, and, as experience has shown, more frequently in *men* than in women.

Among the *factors contributory* to infection **exposure to cold and traumatism** especially may be mentioned. The disorder is therefore encountered with especial frequency in northern climates with marked variations in weather, on the banks of mountainous seas with frequent and persistent fog, and in the spring and autumn, that is, though scarcely exclusively, the exciting inflammatory agents gain entrance into the general blood-stream and lymphatic stream, and thence into the joints through the *tonsils*. In isolated instances *infection* has been observed to occur through *personal association*, as, for instance, in hospitals among those in adjacent beds.

The disorder occurs at times in isolated instances *sporadically*, and at other times *epidemically*. Occasionally it is confined to a few houses—*endemic*—as, for instance, to a single barracks. Recovery from one attack of rheumatism not only does not confer protection, but rather is followed by *increased predisposition* to subsequent attacks, and there are many patients who throughout life suffer from frequent recurrence of the disorder.

Symptoms.—Nothing of a definite nature is known with regard to the *period of incubation of acute articular rheumatism*, although from personal experience I should be led to believe that this is often but a few hours. *Prodromes* occasionally are wholly wanting, or consist in symptoms of catarrhal or lacunar angina. Sometimes the disease sets in with a single chill, or with **repeated chilliness**, which is followed by elevation of temperature to 39° or 40° C. (102.2° or 104° F.), rarely above. It is a peculiarity that many patients **sweat** freely and persistently during the continuance of the fever, so that the skin often is covered with innumerable **miliary vesicles**. The urine, therefore, is generally diminished in amount, highly concentrated, and deposits a considerable sediment of urates (brick-dust sediment). The patients not rarely become exceedingly pale within a short time, and a diminution in the number of red blood-corpuscles, and an increase in the number of colorless corpuscles, will be found. Occasionally **enlargement of the spleen** can be demonstrated.

The **alterations in the joints**, however, are alone decisive in the diagnosis. The affected joints are extremely painful on the slightest pressure and on active and passive movement. They appear thickened and swollen, and on palpation fluctuation can frequently be elicited over the large joints, because fluid exudate in large amount has accumulated in the articular cavity. The skin over the affected joints is not rarely reddened, smooth, glistening, tense, feels hot, and exhibits pitting on pressure, [illegible] in consequence of periarticular edema.

[illegible] described are but rarely confined to a single joint (monarthritis); generally numerous joints are involved. The large joints of the extremities (ankle, knee, wrist, elbow, shoulder) are attacked with especial preference, and this is explained by the circumstance that in walking, standing, and in various manipulations these parts are subjected with especial readiness to mechanical injury. In some instances scarcely a joint is free from alteration, and even the vertebral articulations, those of the lower jaw, the arytenoid cartilages, and the ribs and the symphysis pubis may be involved. It is noteworthy that the articular alterations are most variable and fugacious. Some joints are restored to the normal, while others are attacked; then the changes in the latter disappear, and the former are again affected. If numerous joints are involved, the patient may fall into a most helpless condition. He is no longer able to move his arms and legs, is unable to roll from one side of the body to the other, and requires extraneous aid in dressing, eating, and in every other needed manipulation.

Nothing is known in advance with regard to the *duration of an attack of acute articular rheumatism.* In general, the disease pursues a *subacute course*, extending over from four to eight weeks, although occasionally it may last for a much shorter, or even a considerably longer time. *Relapses* occur with extreme frequency, and especially if the patient arises from bed too early and makes attempts at walking.

Among the *anomalies of acute articular rheumatism* the *larval varieties* should especially be mentioned. *Neuralgia*, *pleuritis*, and *pericarditis* are occasionally observed to occur at times when acute polyarthritis prevails in a considerable number of cases, and they subside so quickly under treatment with salicylic acid, the specific for acute polyarthritis, that the conclusion cannot be avoided that the conditions named are dependent upon the cause of polyarthritis. Occasionally, although the joints remain free, *acute polytendinitis* or *polysynovitis* develops instead.

The *complications* of acute articular rheumatism are exceedingly numerous, *cardiac complications* being the most frequent. It should, therefore, be the rule for the conscientious physician daily to examine the heart most carefully. Most frequently, almost in half of the cases, acute endocarditis occurs, generally involving the leaflets of the mitral valve, and giving rise to permanent insufficiency of the valve, less commonly to stenosis of the mitral orifice. Pericarditis occurs much less commonly than endocarditis, and myocarditis occurs even less frequently. Occasionally, however, pericarditis, myocarditis, and endocarditis are encountered in association. Pleuritis, peritonitis, and meningitis are among the less common complications. Febrile albuminuria occurs frequently; acute nephritis rarely. Inflammatory complications, may however, occur also in many other organs. Occasionally the bodily temperature attains an exceedingly high level, hyperpyretic temperatures resulting, which may within a short time be attended with coma, paralysis of the heart and the brain, and death.

Among the *sequelæ valvular disease of the heart* especially should be mentioned. Not rarely *stiffness of the joints* persists. Occasionally *psychopathy* develops. *Chorea* also should be mentioned among the sequelæ, occasionally occurring even as a complication.

Diagnosis.—The recognition of acute articular rheumatism is easy, because the changes in the joints give rise to striking deformities and impairment of function. The disease might at times be confounded with **secondary acute polyarthritides**, such as occur in the sequence of certain infectious diseases, particularly gonorrhea, scarlet fever, dysentery, and septicopyemia. The history and the conditions present (gonorrheal discharge, exanthem, etc.) are decisive in this connection. Acute articular rheumatism is distinguished from **gout** by the fact that the latter generally begins in the joint of one great toe and is unattended with fever. The diagnosis of larval polyarthritis is more difficult, depending almost upon diagnostic instinct, and being rendered probable by the rapidly curative effect of the salicylates or their substitutes.

Prognosis.—Acute articular rheumatism but rarely terminates fatally, and the *prognosis* is therefore **favorable.** The conditions are, however, rendered more serious from the circumstance that *incurable sequelæ*, particularly valvular disease of the heart, persist, and that the disease often *recurs.*

Anatomic Alterations.—On opening the diseased **joints** scarcely any alterations are occasionally observed. In some joints the articular fluid is increased in amount, is flocculent and turbid, and occasionally almost purulent. The articular capsule and the cartilage are actively injected, the capsule and the villi thickened, and occasionally erosions of the articular cartilage are present. The **muscles** are dry and red like ham, and on microscopic examination exhibit granular, fatty and waxy degeneration. The **heart** is frequently conspicuous for its flabbiness, and exhibits the same histologic alterations as the voluntary muscles. The **spleen** is increased in size and is soft in consistence (infection-spleen). The liver-cells, the epithelial cells of the convoluted uriniferous tubules and the glandular cells of the gastric and intestinal mucous membrane are involved in granular turbidity and fatty degeneration.

Treatment.—Acute articular rheumatism is one of the few infectious diseases for the relief of which **specific treatment** is known. Only exceptionally does salicylic acid or **sodium salicylate** fail:

℞ Sodium salicylate, **1.0 (15 grains).**
Make 20 such starch-capsules

In general, sodium salicylate is better borne than salicylic acid, particularly if the remedy is not taken into an empty stomach, and its administration is preceded by the ingestion of some milk. If, however, nausea, epigastric pain and anorexia cause withdrawal of the remedy, it should be borne in mind that the *salicylates* may be administered also in *the form of enemata* (5.0—75 grains, with 100—3 ounces, of tepid water and 10 drops of tincture of opium once daily). The application of *salicylated ointment* also is to be warmly recommended, the painful joints being massaged therewith morning and evening, and then being enveloped in salicylated cotton:

℞ Sodium salicylate,	
Wool-fat,	
Lard,	each, 30.0 (1 ounce).—M.

To be used morning and evening by inunction.

The internal administration of the salicylates should be immediately suspended on the appearance of the first symptoms of intoxication. These include principally impaired tactile sensibility upon the scalp, a frothy sensation upon the tongue, and roaring in the ears. In addition, there may be mentioned salicylic-acid delirium, salicylic-acid dyspnea, salicylic-acid hematuria, and salicylic-acid exanthemata, the last of which are at times suggestive of measles and at other times of scarlet fever, but which occasionally also are hemorrhagic, vesicular, or pustular in character. Most patients tolerate from 5.0 to 10.0 (75 to 150 grains) of salicylic acid, and some even more, before the first symptoms of intoxication appear. The use of the salicylates should not be resumed until the toxic symptoms have wholly disappeared. During this interval it is well to administer the *substitutes* for salicylic acid, among which **phenacetin** and **antipyrin** are to be preferred:

℞ Phenacetin,	1.0 (15 grains).
Make 10 such powders.	
Dose: 1 powder every three hours.	

℞ Antipyrin,	1.0 (15 grains);
Sugar,	0.5 (7½ ").—M.
Make 10 such powders.	
Dose: 1 powder four times daily.	

It is important to continue the use of the salicylates for as long a time as possible, even after articular swelling and pain have disappeared, in order to avert relapses. Under such circumstances the dose naturally will be gradually reduced, until finally only 1.0 (15 grains) is given thrice daily.

Some experiments in blood-serum therapy have as yet not yielded satisfactory results.

That a patient with acute polyarthritis should remain in *bed* is a matter of course. As long as the fever continues, a **liquid diet** only should be permitted, preferably of milk principally. Severe thirst can be relieved by means of **lemonade.** The use of *substitutes for the salicylates* is justifiable only in the intervals in which symptoms of salicylic-acid intoxication are present, or in those rare instances in which the salicylates are not useful. The administration of phenacetin (1.0—15 grains—thrice daily) or of antipyrin (1.0—15 grains—four times daily) would then be advisable, although salol (1.0—15 grains—every two hours), salipyrin (1.0—15 grains—every two hours), salophen (1.0—15 grains—every two hours), lactophenin (0.5—7½ grains—every two hours), kryofin (1.0—15 grains—thrice daily), and some other remedies have also been recommended.

Occasionally pain and swelling persist in a few joints, or in a single joint. Under such circumstances resort may be had to an **ice-bag, massage, inunctions of an ointment of potassium iodid** or of **iodoform, hydropathic affusions, leeches, cantharidal plaster, faradization,** and the like. Often speedy results will be obtained from the application of a **plaster-bandage,** although this is not rarely followed by rapid wasting of the muscles attached about the joint, and which is to be overcome by massage and faradization.

CHRONIC ARTICULAR RHEUMATISM.

Etiology.—Chronic articular rheumatism is frequently a sequel of acute articular rheumatism, a number of joints never becoming wholly free from pain and swelling. The disorder, however, also occurs independently, especially in elderly persons, and its development is favored by living in cold and damp rooms. Nevertheless, only a contributory influence is to be conceded to the action of **cold** in this connection also, and **bacteria** are to be looked upon as the actual exciting factor of the disease, although their identity has not as yet been established. In addition to exposure to cold, **traumatism** and **over-exertion** favor the development of the disorder.

Symptoms.—Chronic articular rheumatism generally pursues an afebrile course. Occasionally, it is true, transitory febrile movement occurs, particularly if many joints are involved simultaneously or in quick succession. At times the symptoms are confined to **articular pain,** which occurs either only upon touch and movement, or, particularly during the night, without appreciable cause, and radiates for a considerable distance into the affected member. Generally the **joints are enlarged,** although the condition often is one rather of permanent thickening of the articular extremities and the articular capsule, than of an accumulation of fluid exudate within the articular cavity. Movement of the extremities is often attended with palpable and audible grating, because the articular surfaces have become rough and dry. The changes in the joints do not exhibit the sudden variations that are observed in acute articular rheumatism, but rather have a tendency to become localized and to persist for years, or even throughout the whole of life. Periods of greater and of lesser discomfort, it is true, often alternate, the symptoms being aggravated especially during the variable months of autumn and spring, and after physical exertion.

Complications develop much less commonly than with acute articular rheumatism. Endocarditis relatively occurs most frequently. Among the *sequelæ*, in addition to *valvular lesions of the heart*, *rigidity of the joints*—*ankylosis*—especially is to be mentioned, and this is not rarely associated with *muscular atrophy*, particularly of the extensors.

Diagnosis.—The diagnosis of chronic articular rheumatism is, as a rule, not attended with much difficulty. The disorder is distinguished from **deforming arthritis** by the fact that it may occur also in early life, and is generally preceded by attacks of acute articular rheumatism. In contradistinction from **chronic articular gout**, chronic articular rheumatism is characterized by the absence of urarthritic tophi and of increased formation of uric acid.

Prognosis.—Chronic articular rheumatism is an obstinate and troublesome disorder, which, if extensive in distribution, makes a cripple of the patient, but it is, as a rule, unattended with immediate danger to life.

Anatomic Alterations.—The articular cavity is not rarely characterized by excessive dryness. The articular capsule and the synovial villi are generally thickened, and not rarely intracapsular adhesions have formed. The articular surfaces are distorted, often eroded, and the seat of hyperplasia and prominences particularly in the peripheral portions.

Treatment.—In the treatment also of chronic articular rheumatism **salicylates** should at first be employed, although rapid and certain results are not to be expected. The substitutes for the *salicylates* mentioned on p. 354 will generally yield even less. Other **antirheumatics** also have been employed, among which potassium iodid (5.0 : 200—75 grains : 6½ fluidounces ; 15 c.c.—1 tablespoonful—thrice daily), tincture of aconite, and tincture of colchicum (10 drops thrice daily) may be mentioned. The **local treatment** for the joints is the same as that for acute articular rheumatism. Recently local *hot-air baths* have been recommended. Well-to-do patients may be advised during the cold months to avoid the variations in temperature of northern climates, and to visit **climatic health-resorts** in the south (Riviera, Egypt). During the summer **saline, sulphurous, mud, peat,** and **indifferent thermal baths** may be used. Also, **Russian, Irish, Roman,** and **sand baths** yield favorable results in many cases. To overcome stiffness of the joints **massage** and **Swedish movements** may be employed. It is important for the patient to avoid the effects of cold and wet and to wear woollen underclothing.

MUSCULAR RHEUMATISM.

Etiology.—The disorder designated muscular rheumatism is, in all probability, also nothing more than an *infectious disease*, although living infectious agents have as yet not been isolated. The **influence of cold,** which often is considered by the laity as the exciting cause, probably constitutes only a contributing factor. Like other infectious diseases, muscular rheumatism also not rarely occurs in many persons at the same time—thus in *epidemic distribution*, particularly in the variable months of autumn and spring. *Adults* are generally attacked by the disease, which is characterized by an unmistakable tendency to *recurrence.*

Symptoms, Diagnosis, Anatomic Alterations, Prognosis, and Treatment.—The principal symptom of muscular rheumatism is **muscular pain—myalgia**—which occurs spontaneously, and is generally worse principally at night, but which may be induced also by pressure and by movement. Objective alterations are generally wholly wanting. Sometimes but a single muscle is involved, at other times several muscles; or the pain may jump from one muscle to another. In cases of extensive muscular rheumatism motility and functional activity are greatly impaired, and the patients are sometimes compelled to remain in bed for some time. Rheumatism of the thoracic muscles may so greatly interfere with respiration as to give rise to shortness of breath, cyanosis, and not inconsiderable dyspnea. Rheumatism of the muscles of the neck may cause deflection of the head—rheumatic torticollis. Extensive rheumatism of the muscles of the back will give rise to rigidity and immobility of the vertebral column, occasionally with the development of marked curvature forward, so that the clinical picture of tetanus is suggested. At times especially the lumbar muscles upon one or both sides are the seat of such severe pain that the patients are unable to turn or to stoop, and they groan aloud. This condition has been designated *lumbago.*

In accordance with the *duration* of the disease a distinction is made between *acute* and *chronic muscular rheumatism*, and between which subacute rheumatism may be placed as an intermediate stage. Acute muscular rheumatism is occasionally attended with slight febrile movement and profuse sweating, while chronic muscular rheumatism, on the other hand, is unattended with fever. Occasionally endocarditis and myocarditis are associated with the latter as *complications. Muscular induration* may occur as a sequel; that is, dense connective-tissue thickening of the muscle, causing shortening. Care should be taken, further, to avoid confounding rheumatic muscular induration with trichinous muscular changes. The differentiation can be made readily and certainly by excising a small piece of muscle and examining it microscopically.

Scarcely anything of a definite nature is known with regard to the *anatomic alterations* of muscular rheumatism, because opportunities for post-mortem examination are wanting.

The *prognosis* is favorable, as there is scarcely any danger to life, although chronic muscular rheumatism is one of the most obstinate and often incurable diseases.

The *treatment* is the same as that for acute and chronic articular rheumatism. Often speedy and successful results are obtained with electricity.

III. INFECTIOUS DISEASES ATTENDED WITH LOCAL ALTERATIONS IN THE BLOOD AND THE BLOOD-GENERATING ORGANS.

RELAPSING FEVER.

Etiology.—Relapsing fever constantly prevails in *endemic distribution* in some countries of Europe (Ireland, Russian Poland). From these places the pestilence is occasionally carried to other countries, either through vagrants, peddlers, or emigrated laborers. Cases of relapsing fever have from time to time occurred with especial frequency in the larger cities of northern Germany, as, for instance, Königsberg, Dantzic, Berlin, Braunschweig, Magdeburg, Hamburg, etc., and often visiting patients have been the cause for the occurrence of an *epidemic of relapsing fever.* Irish emigrants have in a number of instances been the means of conveying the disease to America.

Infection may take place through *personal association,* through *inanimate objects,* through the *air,* or through the intermediation of a *third person.* It undoubtedly occurs most frequently through personal association or domestic articles. Generally epidemics of relapsing fever are spread from low-class lodging-houses or jails —that is, from places in which roving people of doubtful repute are likely to be found. The bedding, which in such places is but rarely changed, is an especially favorable source of infection, so that many a traveller, in addition to the longed-for rest, finds himself infected if the bed he has used had been previously occupied by a patient suffering from relapsing fever. The dissemination of the disease is further materially favored by the fact that patients suffering from relapsing fever are still for a time capable of travelling in spite of their infectiveness. The conditions indi-

ake it comprehensible that relapsing fever is generally
e of the *lower classes*, occurring especially in the autumn
nter, when lodging-houses are well patronized. In conse-
of the conditions described *men* are more frequently attacked
apsing fever than women, and the disease occurs principally
ults. Recovery from one attack is almost always followed
ermanent *immunity*.

The *infective agent* is contained in the blood, as inoculation-
eriments with the blood have been successful in human
ngs. Although it is probable that the spirilla of relapsing
ver, which are unexceptionally found in the blood during the
brile period, constitute the infectious agents, nevertheless the
lood remains infective after the contained relapsing spirilla are
purposely destroyed, so that probably spores, which hitherto it has
not been possible to demonstrate, constitute the actual agents of
infection. The channels through which the infected materials gain
entrance into the body have not as yet been discovered.

Symptoms and Diagnosis.—The *period of incubation* of
relapsing fever is from five to seven days. *Premonitory symptoms*
—prodromes—often are wholly wanting, although some patients
complain of general malaise, anorexia, and an indefinite sense of
illness. As a rule, the disease sets in with a single chill, followed
by a febrile period of an average duration of a week, with eleva-
tion of temperature to 39° or 40° C. (102.2° or 104° F.), and
frequently even above. In the course of seven days the temper-
ature declines by crisis, with profuse sweating. The patients are
now again free from fever and well for a period averaging seven
days, when a renewed chill occurs, and continued fever sets in,
and only after the new febrile paroxysm terminates by crisis in
from five to seven days do most patients recover permanently.
In some cases, it is true, a third and a fourth febrile recurrenc
take place, the apyretic intervals, as well as the febrile period
being of shorter duration. As may be understood, the course
the temperature of relapsing fever is so distinctive (Fig. 61) th
it is possible to make the diagnosis from this alone. A sim
temperature-curve is known to occur only in some cases of *pse*
leukemia, although the latter is unattended with the blood-
that is distinctive of relapsing fever.

During the febrile period **spirilla of relapsing fever—spiro**
of Obermeier—are found unexceptionally in the blood, and n
ful physician will, at the present day, diagnose relapsing
without demonstration of the spirilla in the blood. In mal
examination it is only necessary to remove a drop of bl
the cleansed tip of a finger by puncture with a needl
make microscopic examination with a magnification of
to 500. Unless one be skilled he will do well to exa
carefully for some time especially those places in the bl

ration where red or colorless corpuscles are apparently engaged in purposeless movement, as this is often due to the actively moving relapsing-spirilla, which displace the blood-corpuscles that are in their way.

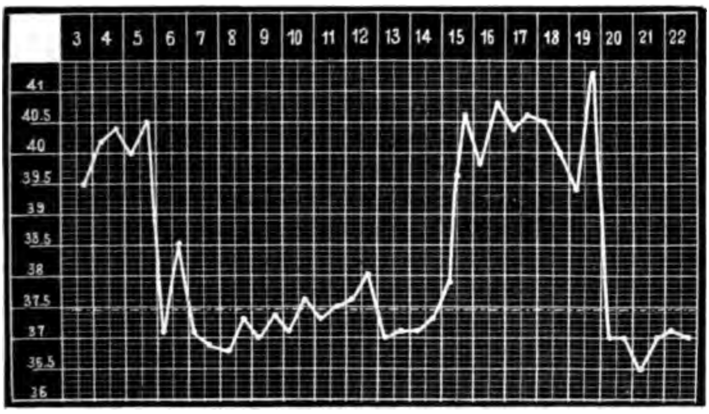


Fig. 61.—Temperature-curve from a case of relapsing fever, with a single relapse (personal observation).

The spirilla of relapsing fever are spiral, filamentous structures, in which from five to eighteen turns can be distinguished (Fig. 62). They attain a length of from 16 to 40 μ (one μ equals 0.001

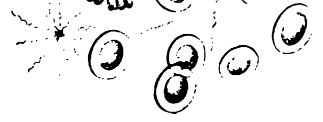


Fig. 62.—Blood from a case of relapsing fever containing spirochetæ of Obermeier; immersion; magnified 1150 times.

mm.) and they shoot across the field of vision often with great activity, exhibiting movement from before backward, and the reverse, lateral twisting and rotation about their longitudinal axis. They are extremely sensitive to all protoplasmic poisons, as a result of the action of which they lose their power of movement. With the occurrence of the crisis of the fever they generally disappear suddenly from the blood, to reappear with the succeeding febrile paroxysms. Occasionally, it is true, they may also be encountered in the blood for a few days after the crisis, with gradual diminution in motility. It has hitherto not been possible to cultivate them artificially. In hermetically sealed capillary tubes they retain their power of movement in the blood for several days, after which they lose this power and disintegrate into granular masses.

Individuals with relapsing fever complain frequently of severe pulsating **headache** and of such marked **vertigo** that they are compelled to remain in bed. The **face** presents a sallow appearance. The **liver** and the **spleen** are enlarged and generally tender on pressure. In addition, the manifestations of fever are present, particularly increased thirst, a coated tongue, and anorexia. The urine is scanty, high-colored, and frequently contains albumin (febrile albuminuria). The occurrence of the crisis is not rarely preceded for a short time by symptoms of a **critical perturbation,** as disclosed in unanticipated chill, unusual elevation of temperature, and delirium, and which are likely to persist but for a short time (from one to three hours). They are without serious significance. On the termination of the crisis the patients improve with almost remarkable rapidity, and in the course of a few days are able to get out of bed again.

Complications not rarely attend relapsing fever, those referable especially to the spleen and the liver being distinctive. The presence of cardiac-systolic *vascular murmurs* over the *spleen* is not significant. Symptoms of *perisplenitis* (perisplenitic friction-murmur, pain), and still more those of *abscess of the spleen,* are worthy of greater consideration. The existence of the latter complication will be indicated—in addition to pain over the spleen and enlargement of the spleen—especially by long-continued fever, chills and sweats, the fever not rarely exhibiting an intermittent character. Occasionally abscess of the spleen ruptures suddenly into adjacent structures, as, for instance, the pleura, the bronchial tubes, the stomach, the intestines, or the urinary passages, the peritoneal cavity, or externally. Some of these occurrences are almost necessarily fatal, particularly rupture into the abdominal cavity, which is usually complicated by fatal peritonitis. *Rupture of the spleen* has been observed in a number of cases of relapsing fever.

Bilious relapsing fever results in consequence of profound disease of the liver.[1] It is known also as *bilious typhoid,* although it is well to avoid this designation, as it has obviously been applied to various morbid conditions. Patients with bilious relapsing fever exhibit marked jaundice of the skin and mucous membranes. The urine also acquires a deeply icteric hue, generally becomes diminished in amount, and contains, in addition to biliary coloring-matter, albumin and tube-casts, and often also tyrosin-needles and leucin-spheres. Erythema and urticaria often appear upon the skin, and finally symptoms of blood-dissolution (hemorrhagic diathesis) make their appearance. Smaller and larger hemorrhages occur beneath the skin, and extravasations of blood take place from various mucous membranes, thus, for instance, bleeding from the gums, epistaxis, hematemesis, enterorrhagia, hematuria, etc. The patients fail rapidly, become unconscious and delirious, and succumb frequently within a few days from excessive exhaustion. The liver is generally exceedingly sensitive to pressure, and increases considerably in size. The clinical picture resembles that of acute yellow atrophy of the liver, and is probably dependent upon a hepatogenous auto-intoxication that has been designated also cholemia.

---

[1] This is a symptomatic disorder of varied origin having nothing to do with relapsing fever, and is not due to the activity of the spirillum peculiar to that disease. It may be compared to the so-called hepatic intermittent fever resulting from the presence of suppuration or other irritation within the hepatic ducts.—A. A. E.

Prognosis.—The prognosis of relapsing fever is not unfavorable so long as complications are absent. Drunkards especially are exposed to great risk.

Anatomic Alterations.—The dead body generally presents a slight degree of **icterus.** The **viscera** exhibit alterations such as are observed in other acute infectious diseases (dry, ham-colored muscles, with granular turbidity and fatty or waxy degeneration of the muscle-fibers, granular turbidity and fatty degeneration of the fibers of the heart-muscle and of the glandular cells, enlargement of the spleen and the liver). It is distinctive for the **follicles of the spleen** to be enlarged, and often to be transformed into necrotic or purulent foci. In the **bone-marrow** also necrotic and purulent foci are not rarely demonstrable. In the venous spaces of the spleen and the bone-marrow relapsing spirilla are encountered, occasionally in a convoluted arrangement. The spirilla of relapsing fever retain their motility in the dead body for a few hours, and the cadaver is therefore infective.

Treatment.—The treatment of relapsing fever is the same as that for typhus fever (pp. 314–316). **Blood-serum therapy** has been attempted, but has not yet yielded encouraging results. In cases of bilious relapsing fever quinin (2.0 : 30 grains) has been recommended.

MALARIAL FEVER (SWAMP-FEVER).

Etiology.—Malarial diseases are justly designated *swamp-fever*, because they occur especially in marshy localities. The swampy regions of Italy, Hungary, and Greece, and the tropics are therefore notorious as breeding-places for malarial diseases. The *mouths of large rivers,* which often slowly empty into the sea through several arms, are comparable with marshy regions. It is therefore not surprising that malarial diseases are quite widespread along the Memel, the Pregel, the Vistula, the Oder, the Elbe, and the Rhine, as well as along the Danube, and the large rivers of the south European peninsulas. Also the *shores of the sea,* particularly seas with ebb and flow, possess the characters of swampy regions, and are therefore sources of malarial disease. Malarial diseases are generally encountered also in *peat-regions* (Hanover, East Friesland). Frequently the *banks of inland seas* were sources of malaria, and the Swiss lakes also were formerly considered malarial, although they have for many years been freed from this disease by proper drainage.

At times certain regions become *temporarily malarial.* Especially inundations, earthquakes, destruction of forests, erection of fortifications, and excavations are not rarely followed immediately by cases of malaria. Vegetable decay and neglect in the cultivation of the soil favor the development of malaria, while reverse conditions will cause the disappearance of the disease. Occasionally *accidental foci of malaria* are created, as, for instance, by the toleration of pools of water in the neighborhood of inhabited houses, or on ships from imperfect removal of bilge-water. The conditions for the development of malarial diseases are therefore present wherever water of not too considerable depth stagnates. Only those persons are attacked by malaria who visit a malarial locality. The infectious material is thus contained in the earth, and is conveyed through the air for only a short distance. To this peculiarity the designation *miasm* has been applied, and the malarial diseases have been included among the *miasmatic infectious diseases.* If infection from one person to another occurs at all, it takes place in any event with extreme rarity. Some experiences indicate that the malarial poison possesses a certain weight and difficulty of movement, for it has been observed that it does not generally reach the upper stories of a house, and that walls of moderate height constitute an insuperable barrier to its diffusion in the vicinity. In non-marshy regions malaria generally occurs *sporadically.* The patients are then usually individuals who have come from malarial regions. Also in marshy regions malaria is at times especially prevalent—*epidemic.* Such epidemics occur particularly in the late summer and autumn.

The *infectious agent of malaria* consists in the **plasmodia** discovered in the blood in 1880 by Laveran, and which are present in the blood principally, and occasionally even exclusively, at the time of the febrile paroxysm. These are not bacteria, but *protozoa,* and thus animal organisms. Further, opinions as to their precise

At the time of the febrile paroxysm *malarial plasmodia* gain entrance into the red blood-corpuscles, in which they first appear as small colorless, roundish bodies, engaged in active movement, and, like amebæ, frequently undergo change in shape. The originally small bodies increase progressively in size, and at the same time brownish and blackish spherical (iron-free) granules appear in them, which move actively to and fro. The greater the number of the latter that are visible, the paler and the more deficient in coloring-matter does the red blood-corpuscle become, so that it is probable that the granules are formed from the hemoglobin of the red corpuscle by the malarial plasmodia. Eventually the plasmodium has become so large that it not only fills the red blood-corpuscle, but often is even larger. This can happen only by the red corpuscle itself becoming distended and enlarged (Fig. 63). Processes of multiplication and division now take place in the malarial plasmodia. Some plasmodia, naturally, first rupture the red blood-corpuscle and become free before they undergo division. In the process of *division* the pigment-granules collect at the center of the plasmodium, while the remainder of the body becomes constricted off as rather a spherical or oval structure, giving rise to the appearance of a sunflower or a daisy (Fig. 63, *g*). The constrictions separate from one another and form unpigmented young plasmodia, which with the advent of the next febrile paroxysm gain entrance into the red blood-corpuscles and again bring about in these the changes that have been described. The pigmented residuum is taken up by colorless blood-corpuscles and in part deposited in other viscera.

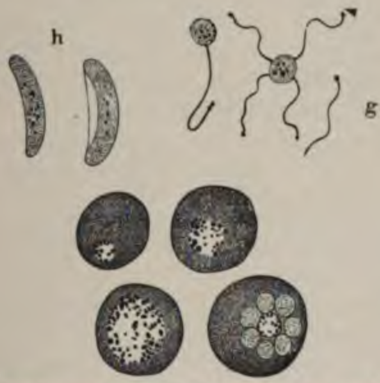


FIG. 63.—Gradual development of malarial plasmodia: *g*, flagellated varieties and free flagella; *h*, crescentic varieties; magnified 1000 times (personal observation, Zurich clinic).

In addition to the varieties of plasmodia already described, *flagellated* and *crescentic varieties* occur. The *flagellated varieties* (Fig. 63, *g*) are motile plasmodia, free in the blood, provided with one or several long flagellated processes, with the aid of which they engage in active vermicular movement. The free extremity of the flagellum generally exhibits a small bulbous enlargement. Here and there flagella become detached from the body of the plasmodium, and may be seen in the blood as independent motile, wavy structures. Nothing of a definite nature is known with regard to the significance of the flagellated varieties. By some they are considered plasmodia at the height of their development, and by others as degenerated forms. The same statement is applicable also to the *crescentic bodies* (Fig. 63, *h*), concerning which it is known only that they are present in the blood especially in cases of malignant tropical malarial fever. Often a sort of membrane is adherent to them.

To the different varieties of malarial fever, especially the intermittent, correspond also different varieties of plasmodia, which can be differentiated by the trained eye with the aid of the microscope. Biologically they are distinguished from one another by the fact that their periods of maturation occupy varying lengths of time. Thus, in cases of tertian intermittent the period of maturation occupies forty-eight hours; in cases of quartan intermittent, seventy-two hours. Quotidian intermittent results from the presence in the blood of plasmodia of tertian fever with two different periods of maturation, although special varieties of plasmodia are believed to be responsible for some cases of quotidian fever.

Although it has, as yet, not been possible to secure pure cultures of malarial plasmodia, and to inoculate these successfully upon animals or human beings, it has, nevertheless, been demonstrated by inoculation-experiments upon human beings that the infectious material is contained in the blood. Similar bodies have, further, been found also in birds, tortoises, and lizards. The medium through which malarial plasmodia gain entrance into the human body is not yet known with certainty, although the air, water, and insects (mosquitoes) have been held responsible.[1]

Occasionally malaria and some other infectious disease—as, for instance, typhoid fever, relapsing fever, or dysentery—have occurred simultaneously in the same person. In the tropics malarial fever and dysentery often occur endemically at the same time.

Symptoms and Diagnosis.—Malarial diseases are attended with various manifestations, and accordingly intermittent fever, larval intermittent fever, pernicious or comatose fever, continuous and remittent fever, and the malarial cachexia have been distinguished. The *period of incubation* varies between hours and several months. Progressive pallor and increasing languor frequently occur as prodromes.

Intermittent fever is the variety of malaria that occurs most frequently in non-tropical countries. The disease is characterized by febrile paroxysms of several hours' duration, which recur at regular intervals, and pursue a definite course. The febrile paroxysm sets in with a severe *chill*, which occasionally is preceded by a sense of languor, exhaustion, and marked pallor of the skin. The teeth chatter, the body is agitated, complaint is made of a sense of icy coldness in the extremities, and the body also feels cold. Obviously, the cutaneous vessels are in a state of spasm, as the skin appears pale, only a small amount of dark-red blood escaping from a needle-prick, and the turgor of the skin is diminished to such a degree that finger-rings, for instance, become too large and slip from the fingers. The eyes are deeply sunken and surrounded by blue rings. Often active yawning occurs. If the temperature is taken in the rectum, it will be readily discovered that the internal temperature of the body is already elevated during the period of chill, and that an unusually marked disparity exists between the cutaneous and the internal temperature of the body. In the course of from half an hour to two hours the stage of chill is replaced by the *febrile* or *pyretic stage*. The patient is conscious of a feeling as if an increasing amount of heat were being constantly diffused from within outward, and as if the disagreeable sense of coldness were subsiding, until finally it is replaced by a sense of burning, almost destructive heat. At the same time the appearance of the patient undergoes a change.. The early pallor disappears, and is replaced by a vivid febrile redness. The previously cold skin feels dry and hot, and its turgor is restored. The pulse becomes accelerated and full, and the patient often complains of distressing beating in the head. He suffers from headache, and often, also, from vertigo. Thirst is marked and appetite is deficient. The blood contains malarial plasmodia. Of especial importance is considerable *enlargement of the spleen*. At the same time the spleen is frequently tender on pressure, and occasionally is the seat of cardiac-systolic vascular murmurs audible on auscultation. The *liver*, also, is generally somewhat enlarged and tender on pressure. The urine is passed in small amount, and is high-colored, both in the period of chill and in that of fever. Not rarely slight febrile albuminuria is present. After the febrile period has lasted on an average for from four to six hours it is followed by the sweating-stage.

---

[1] There is no longer any doubt that malaria is definitely conveyed through mosquitoes (especially, if not exclusively, the variety Anopheles), which are considered as the definite, while human beings are looked upon as the intermediate hosts for the plasmodia. The disease is associated with stagnant water, heat, and decomposing vegetation, because these favor the presence and activity of mosquitoes. On the other hand, the evidence is not sufficiently strong to exclude altogether the possibility of transmission through air and water.—A. A. E.

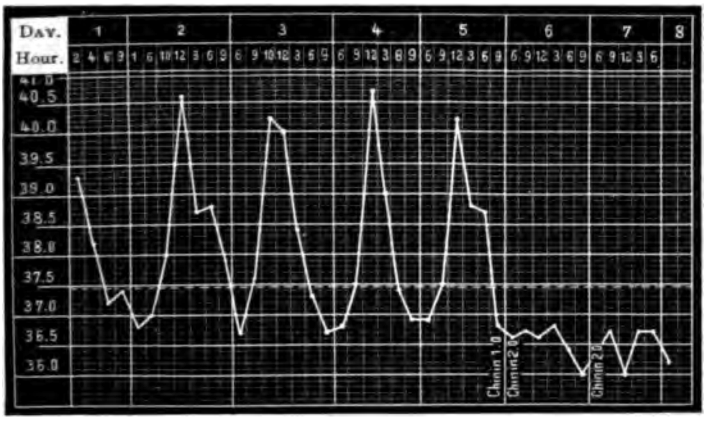


FIG. 64.—Temperature-curve from a case of quotidian intermittent fever, in a Piedmontese, 33 years old (personal observation, Zurich clinic).

The *sweating-stage* is characterized by profuse perspiration. This is often so abundant that the body-linen and articles of clothing are saturated, and an eruption of *miliaria* appears upon the skin. At the same time the bodily temperature declines, and within from two to four hours it will again have reached the normal. The patient continues to feel ill and sore for some time, but generally recovers with marked rapidity. In the interval between two paroxysms the malarial plasmodia either disappear completely from the blood, or they occur in much smaller number, and with slower movement of their entire body and the contained pigment-granulations. The spleen generally becomes somewhat diminished in size, again to become increased in size with the advent of a new febrile paroxysm. Generally the febrile attacks begin by day, and but rarely during the first hours of the night. Should the patient be asleep during the paroxysm the disease may occasionally be unrecognized for a long time.

In accordance with the interval that elapses between two successive febrile paroxysms the *type of an intermittent fever* is distinguished. If a febrile paroxysm occurs every day, the designation quotidian intermittent fever is employed. Tertian intermittent fever is attended with an afebrile day between two febrile days; quartan intermittent fever with an intermission of two days; and, in the same way, quintan, sextan, septan, and octan varieties of intermittent fever are distinguished. Earlier physicians even believed that types with longer intervals occurred. Most patients in the temperate zone suffer from tertian or quotidian intermittent fever. It will be readily understood that the temperature-curve in a case of intermittent fever pursues so distinctive a course that from it alone the disease and its special variety can be recognized. In illustration, reference may be made to Figs. 64 and 65, in which are reproduced the course of a quotidian intermittent fever and that of a tertian intermittent fever.

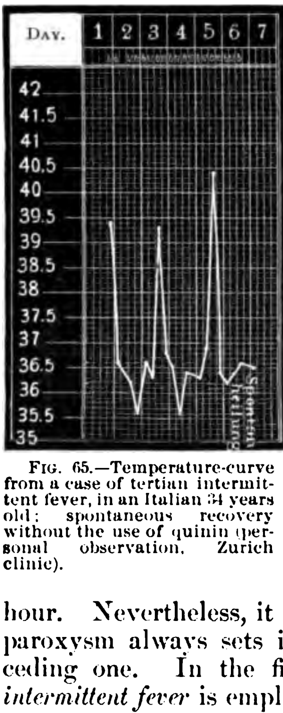


FIG. 65.—Temperature-curve from a case of tertian intermittent fever, in an Italian 34 years old: spontaneous recovery without the use of quinin (personal observation, Zurich clinic).

In the majority of cases the febrile paroxysms constantly recur at the same hour. Nevertheless, it occurs occasionally that each new febrile paroxysm always sets in somewhat earlier or later than the preceding one. In the first instance the designation *anticipating intermittent fever* is employed, and in the latter *postponed intermittent fever*. Naturally it may happen that through anticipation of the febrile period a case of tertian may gradually be transformed into a quotidian fever, and conversely, through postponement, a quotidian may be transformed into a tertian.

Duplicated intermittent fever is a rare condition. In cases of duplicated quotidian intermittent fever two febrile paroxysms occur daily, being repeated at the same hour. In cases of duplicated tertian fever one paroxysm occurs daily, although on alternate days it sets in at a different hour than on the intervening days. In cases of duplicated quartan fever two febrile days are separated by an afebrile day.

The *recognition of intermittent fever* can be readily and certainly made by the demonstration of plasmodia in the blood. If dependence were placed solely upon the temperature-curve, the differentiation from miliary tuberculosis, latent suppuration, and septicopyemic disease (ulcerative endocarditis) would have to be made. Formerly, in the differential diagnosis the physician was frequently restricted to the results yielded by the administration of quinin, which controlled intermittent fever within a short time with the same certainty that it exerted no influence upon the other diseases named.

Larval intermittent fever is characterized by abnormal disturbance in one or another viscus, which, like the febrile paroxysm of a fully developed intermittent fever, is repeated at definite intervals. Occasionally the disturbance is initiated with slight chilliness, is followed by slight elevation of temperature, and disappears with slight sweating. The spleen is palpable and enlarged. Whether the malarial plasmodia are demonstrable in the blood or not is scarcely known. The principal remedy in the treatment of all malarial diseases—quinin—is with certainty useful, also, for the relief of larval intermittent fever.

Larval intermittent fever appears most frequently as an *intermittent neuralgia*, of which branches of the trigeminus, and particularly the supraorbital nerve, are with especial frequency the seat. There may also be intermittent vomiting, gastralgia, cough, asthma, paralysis of the vocal bands, dysuria, diarrhea, vertigo, syncope, paralysis, convulsions, delirium, aphasia, psychopathy, and a number of other conditions.

Without doubt much that has been described as larval intermittent fever has nothing whatever to do with malarial infection. Neuralgia particularly may exhibit an intermittent character without the patient having ever been in a malarial region; and such neuralgia likewise often subsides quickly under the use of quinin. If in the diagnosis the greatest importance be attached to the presence of plasmodia in the blood, or at least to enlargement of the spleen, not much of a definite character remains of larval intermittent fever.

Comatose and pernicious intermittent fever is not a special variety of malarial fever. It is, rather, an ordinary intermittent fever in which some one symptom or another acquires dangerous intensity and not rarely leads to a fatal termination. Thus, the patient may become so cold during the stage of chill that death results with progressively declining bodily temperature—*algid pernicious* *intermittent fever;* or, with the advent of the sweating-stage, the perspiration becomes so profuse and exhausting that death results from exhaustion—*diaphoretic pernicious intermittent fever*. Under the two conditions described the fatal manifestations are in and of themselves peculiar to every case of intermittent fever. It may, however, also happen that quite unusual disturbances may arise (comatose intermittent fever), and that these may compromise the maintenance of life (comatose and pernicious intermittent fever). Occasionally syncope takes place during the chill, and from which the patient may not awaken; apoplectic, epileptic, and tetaniform attacks may occur; alarming bronchitic, asthmatic, or pneumonic symptoms may appear; attacks of palpitation of the heart, pleuritis, pericarditis, peritonitis, gastralgia, diarrhea, jaundice, internal hemorrhage, erysipelas, and the like, may occur. In accordance with the character of the disturbances the designations syncopal, apoplectic, epileptic, tetaniform, bronchitic, asthmatic, pneumonic, cardiac, etc., pernicious intermittent fever are employed.

Remittent and continued fever occurs most exclusively in tropical and subtropical countries. The fever pursues a remittent or a continued type, the spleen is enlarged, generally also the liver, and the blood contains malarial plasmodia, not rarely crescentic forms. Often deep jaundice appears, or hematuria, hematemesis, and enterorrhagia occur. The duration of the disease varies from days to weeks or months. If it be not correctly recognized and treated, death may result from exhaustion.

Malarial Cachexia.—Individuals residing in malarial regions not rarely acquire gradually an unusually pallid, almost earthy-gray color, lose strength, complain of palpitation of the heart and shortness of breath on physical exertion, and exhibit often quite marked enlargement of the spleen and generally also of the liver. Obviously, the malarial virus has interfered with the process of blood-formation, without the previous existence of other varieties of malarial disease. Death occurs in consequence of excessive exhaustion if the malarial cachexia cannot be relieved.

Among the *sequelæ* of malaria two especially should be mentioned—namely, *melanemia* and *amyloid degeneration*. A number of other sequelæ, however, also occur. We shall mention here additionally only chronic nephritis, chronic interstitial hepatitis, and leukemia.

In the presence of *melanemia* brownish and blackish granules and plates of pigment are found in the blood, which are either free and in motion in the blood-plasma, or are enclosed within colorless blood-corpuscles. Occasionally they are aggregated into cylindric or plug-like structures, or enclosed in spindle-shaped cells (endothelial cells of the vessels of the spleen). Obviously the pigment-granules are derived in part from the malarial plasmodia, in the multiplication of which, as has been mentioned, they become free ; but in part they are derived also from the transformation of hemoglobin, which is set free in the disintegration of red blood-corpuscles. Pigment of the latter variety contains iron (hemosiderin). The pigment is carried to the various tissues by the colorless blood-corpuscles, and as a result diverse alterations take place. Its deposition in the cutis and in the lowermost layer of epithelial cells of the Malpighian layer gives rise to a *smoky-gray* and *graphite-like discoloration of the skin*, which will suggest the diagnosis of melanemia even on superficial inspection of the patient. At times profound *cerebral disturbances* occur. The patient becomes comatose or wildly delirious, with clonic convulsions and paralysis. Should death occur, the cerebral cortex will be found to present a grayish discoloration and to be laden with pigment-granules, which are accumulated especially in the blood-capillaries, and in places have formed pigment-emboli. Occasionally *derangement in the functions of the stomach*, the *intestines*, the *liver*, or the *kidneys* occurs, and in all of these organs pigment-granules can be demonstrated. An abundance of pigment will have accumulated also in the spleen, the bone-marrow, and the lymphatic glands. *Amyloid degeneration* generally develops only in the sequence of malaria of long standing that has given rise to marked malarial cachexia.

Prognosis.—The prognosis of malarial fever is good in the case of patients who are able to remove permanently from a marshy region, as only under such conditions is permanent recovery from the disorder possible. In addition, the prognosis naturally varies in accordance with the form of malarial disease. Pernicious, comatose, remittent, or continued malarial fever is a much more serious disease than ordinary or larval intermittent fever. The tropical malarial diseases properly are especially feared, as many an explorer and colonist has succumbed to them.

Anatomic Alterations.—Among the anatomic alterations, especially the condition of the **spleen** is to be taken into consideration, in addition to the changes already mentioned. The spleen is greatly increased in size. In recent cases it is soft in consistency, while in older cases it acquires a dense consistency in consequence of hyperplasia of the trabecular connective tissue. The capsule of the spleen is, in recent cases, occasionally the seat of fibrinous perisplenitis, while in older cases it is thickened. The spleen often contains a large amount of brownish and blackish pigment, which is present in part in the splenic cells and in part in the trabecular connective tissue.

Treatment.—In the treatment of malarial fever **quinin** has proved a reliable *specific*, and it is superfluous to seek for substitutes. Quinin destroys the malarial plasmodia. In cases of intermittent fever it is sufficient to administer 1.0 (15 grains) of quinin hydrochlorate three hours before the onset of the chill :

℞ Quinin hydrochlorate, 1.0 (15 grains).
Make 5 such starch-capsules.
To be taken as directed.

Occasionally the dose must be doubled, so that two doses, each of 1 gram (15 grains), of powdered quinin are taken at an interval of half an hour. Frequently the febrile paroxysm fails to recur after the first administration of quinin, although it is well to continue the quinin on the following days for about a week.[1] In order to expedite the reduction in the size of the spleen an **ice-bag** may be kept constantly applied over this organ. As soon as the spleen is enlarged recurrence is to be feared. For the relief of the anemia iron and arsenic may subsequently be prescribed :

℞ Iron lactate,
Sodium bromid, each, 10.0 (2½ drams) ;
Arsenous acid, 0.1 (1½ grains) ;
Powdered althea-root, sufficient to make 100 pills.—M.
Dose : 2 pills thrice daily half an hour after meals.

Some individuals tolerate quinin badly, being seized, after its administration, with troublesome vomiting, roaring in the ears, occasionally delirium, exanthemata, after large doses even amaurosis. Under such circumstances the drug may be administered in the form of an *enema*, 3.0 (45 grains) of quinin hydrochlorate being mixed with 50 c.c. of tepid water, 10 drops of tincture of opium, and 5 grams (75 grains) of starch being added, and the mixture being slowly introduced into the rectum. The introduction of quinin *beneath the skin* might also be practised. Quinin is soluble in glycerin with the aid of heat. It is, therefore, necessary before using the solution, in which the glycerin has been precipitated in the cold as a white crystalline mass, to apply heat and effect solution by immersion in warm water.

℞ Quinin hydrochlorate, 5.0 (75 grains) ;
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose : 15 minims injected subcutaneously.

In cases in which quinin is not tolerated in any form, **arsenic** should be resorted to, as, for instance :

℞ Solution of potassium arsenite,
Bitter-almond water, each, 5.0 (75 minims).—M.
Dose : 10 drops thrice daily, after meals.

Methylene-blue also has been reported as being still more destructive to the malarial plasmodia than quinin, and it has therefore been recommended in the place of the latter (0.1—1½ grains —every two hours). Quinin and arsenic are also the most efficacious remedies for *larval intermittent fever*. The quinin must not rarely be given in large doses (up to 5.0—75 grains—and even more). In cases of intermittent neuralgia electricity also may be employed with advantage. The *pernicious, comatose, continued,* and *remittent* malarial fevers require prompt and vigorous administration of quinin if the threatened dangers are to be averted. For the *malarial cachexia* quinin is prescribed from time to time in alternation with iron and arsenic in the form mentioned on p. 370.

---

[1] It is often useful to initiate the treatment by the administration of 5 or 10 grains of calomel, followed by a saline laxative.

A paroxysm can sometimes be aborted by subcutaneous injection of pilocarpin hydrochlorate (gr. $\frac{1}{12}$ or gr. $\frac{1}{8}$) or of morphin sulphate (gr. $\frac{1}{8}$ or gr. $\frac{1}{4}$).

Quinin and urea hydrochlorate is a reliable preparation, both by internal and by subcutaneous administration.—A. A. E.

Great significance is to be attached to the *prophylaxis* in the control of malarial fever. With regard to *individual prophylaxis,* such individuals as are compelled to reside in malarial localities should be advised to take constantly small doses of quinin (0.3—4½ grains—daily). In addition, they should avoid being in the open air in the early morning and at dusk, and especially should refrain from sleeping and lying for any considerable length of time upon the ground; the bed-room should not be upon the ground floor, but upon one of the higher floors, and with regard to diet and mode of life they should conform to the rules adopted by the natives as a result of their own experience. Sleeping at an open window is particularly injurious; and the use of fish and acid fruits (melons, peaches) is also warned against. An important factor in bringing about permanent recovery is *permanent removal from a malarial region.*

In order to deprive malarial localities of their infectious properties it has been recommended that marshy regions be rendered dry, or be submerged beneath such a depth of water that penetration of the sun's heat and putrefaction of vegetable matter will be impossible. For the drying up of the marshes the planting of the fever-tree, the Eucalyptus globulus, has been recommended among other things. Occasionally places situated on rivers have been freed from malaria by processes of grading and the construction of dams. By the removal of pools, or, aboard ship, of stagnant bilge-water and other like measures, local malarial foci can be eradicated.[1]

BUBONIC PLAGUE.

Etiology.—The plague is an infectious disease that has been known since most remote times, and which originally appears to have been indigenous in India and farther Asia. In Europe it mmitted great ravages, particularly in the Middle Ages, and an pidemic of the fourteenth century is especially notorious, which as been designated the "black death" because hemorrhage from he lungs occurred with extreme frequency. Occasionally one-fourth of the population has succumbed to the plague. Only since the middle of the nineteenth century has the plague disappeared from Europe, although there is yet always danger of renewed importation from Asia, where, particularly in recent years, extensive epidemics of the disease have again occurred.

---

[1] The important object in the prevention of malaria is the removal of conditions that favor the growth of mosquitoes, and, in failure to do this, to protect human beings against the bites of the insects by means of screens in doors and windows of houses, by shielding the surface of the body and by avoiding exposure at night, when the infecting mosquitoes abound.—A. A. E.

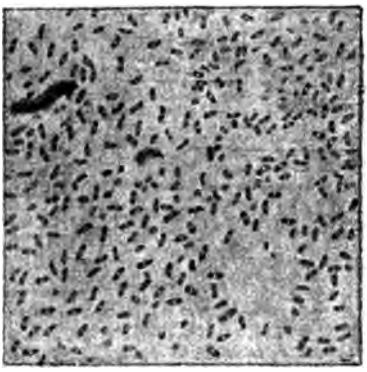


FIG. 66.—Bacillus of bubonic plague (Yersin).

The exciting agent of plague is believed to be the **plague-bacillus** (Fig. 66) discovered by Yersin and Kitasato in 1894—a rod with rounded extremities, which stain more deeply with methylene-blue than the middle. Not only has it been possible to grow plague-bacilli in pure culture, and to inoculate animals successfully with them, but involuntary inoculation of human beings with cultures of plague-bacilli has recently occurred in one of the clinical laboratories at Vienna, as a result of which three persons succumbed. Plague-bacilli are found in the purulent accumulations of the disease, in inflamed lymphatic glands, in the enlarged spleen, occasionally in the blood, and it is said also in large number in the urine and the feces, and, in cases of plague-pneumonia, also in the expectoration.

Plague-bacilli not rarely gain entrance into the human body through small *cutaneous wounds*, particularly fissured wounds of the hands and feet being especially dangerous in this connection. At times the *tonsils* constitute the portal of entry for plague-bacilli. Occasionally *plague-bacilli* are inhaled, and thus give rise to plague. It is still a debatable question whether infection may take place through the stomach and intestines from plague-bacilli that have been swallowed. Infection may take place through immediate *contact with plague-patients*, through *inanimate objects*, through the *air*, or through the intermediation of a *third person*. The dirtier and the more careless the mode of life in a community, the more readily does plague spread. For this reason the disease does not become extinct in the Orient. ***Rats*** appear to play an important *rôle* in the dissemination of the pestilence, are attacked in large numbers during the prevalence of plague, and their number is almost unlimited in the Orient. Some *insects*, also, may convey the infection, as it has been possible to demonstrate the presence of plague-bacilli in the blood of *fleas* that have been removed from rats suffering from plague.

Symptoms.—The *period of incubation* of plague varies from two to seven days. In accordance with the clinical manifestations three varieties of plague have recently been distinguished, and these have been designated *bubonic*, *septic*, and *pneumonic plague*.

Bubonic plague generally begins with a **chill**, which is followed by *elevation of temperature* to 39° or 40° C. (102.2° or 104° F.) and above. Soon the peripheral **lymphatic glands** become the seat of pain and swelling; thus, the inguinal, brachial, axillary, submaxillary, cervical lymphatic glands. The enlargement of the lymphatic glands is frequently by no means symmetric, and some collections of glands are conspicuous on account of the especially advanced inflammatory alterations they present. Some packets of glands begin to exhibit fluctuation in the course of two or three days, and finally rupture externally. In many patients this is followed by material improvement in the general condition. The spleen is almost always enlarged. The general condition may be affected in various ways. Some patients feel scarcely ill, and are unwilling to remain in bed, while others are vertiginous, become comatose, and occasionally die within the shortest possible time from exhaustion and cardiac paralysis.

In cases of *septic plague* either the enlargement of the lymphatic glands pursues the course described, or the alterations in the glands are confined to slight swelling and tenderness. The patients exhibit profound collapse, and, above all, hemorrhages take place from the stomach and the intestines. The disease generally pursues an unfavorable course if plague-bacilli can be demonstrated in the blood.

Plague-pneumonia resembles ordinary pneumonia, although, in addition to pneumonia cocci and streptococci, plague-bacilli also are present in the expectoration.

The *duration of an attack of plague* is from one to two weeks. Among the *complications* parenchymatous *keratitis* especially is to be mentioned, not rarely terminating in suppuration and destruction of the eye.

Diagnosis.—Since the discovery of the *plague-bacillus* the diagnosis of the plague has been possible with certainty. Isolated instances were formerly confounded with especial frequency with syphilis. Plague-bacilli can but rarely be found directly in the blood. It is well to prepare an agar-culture from the blood, in which the plague-bacilli will develop in the thermostat within forty-eight hours, and can be distinguished by their bluish-gray color.

Prognosis.—The prognosis of plague is serious, as in not a few epidemics the mortality has been as high as 90 per cent.

Anatomic Alterations.—Post-mortem examinations show that not only the external, but also the internal, **lymphatic glands** are involved in inflammatory swelling. The **spleen** and the **intestinal follicles** also are enlarged and swollen. On microscopic examination necrotic foci are found in the organs named. In cases of septic plague purulent accumulations are present in various organs, and, in cases of plague-pneumonia, bronchopneumonic or lobar areas of inflammation.

Treatment.—Blood-serum therapy has been recommended as **a specific in the treatment of plague**, although its success has been questioned from various sources. Accordingly, **expectant treatment**, or, in the presence of threatening symptoms, **symptomatic treatment**, must be resorted to. The latter includes the use of *stimulants*. It is doubtful if inunctions with *mercurial ointment* exert any influence upon the enlarged lymphatic glands. Suppurating lymphatic glands should be incised with the knife and be disinfected. Haffkine has recommended *protective inoculations* as a *prophylactic measure*. The future must decide whether these are of practical value. For the present, it is more important to isolate patients suffering from plague, to burn their possessions, or to disinfect them with great care, and also to sterilize the urine, the feces, the expectoration, and the pus. It is important to exercise most scrupulous domestic and personal cleanliness and to insure the destruction of rats.

IV. INFECTIOUS DISEASES ATTENDED WITH LOCAL ALTERATIONS IN THE RESPIRATORY ORGANS.

WHOOPING-COUGH (PERTUSSIS).

Etiology.—Whooping-cough is preëminently an *infectious disease of childhood;* only exceptionally does it occur in adults also. In the latter event it attacks principally women, especially pallid, irritable, and nervous women. **Pregnancy** also appears to constitute an influence predisposing to infection. Further, females seem to predominate among the victims of the disease even in childhood, and this has been attributed to their more readily irritable nervous system.

The *infective agent* of whooping-cough is as yet unknown, and the bacteria and protozoa that have hitherto been found in the sputum of patients lack confirmation. Clinical experience shows that infection may take place through *contact*, through the *air*, through the intermediation of a *third person* and of *inanimate objects*. Most children acquire the infection in walking out of doors, on playgrounds, or in school. The infective material is correctly suspected to be contained in the sputum. As it is customary to advise patients with whooping-cough to spend a good deal of time walking in the open air, and as many patients eject their sputum indiscriminately upon the street, where it readily dries and is converted into dust and is inhaled by healthy individuals, it will be readily understood that whooping-cough, by reason of its often long duration, is peculiarly capable of giving rise to a large number of sources of infection. Most children are attacked between the first and the seventh year of life. Within the first six months of life infants generally remain exempt, possibly because they are little exposed to the air, and then often wear veils. Whooping-cough has, however, been observed at times to occur in the first days of life if the mother had suffered from the disease during pregnancy. Those who have passed through an attack of whooping-cough generally remain exempt from subsequent infection, as a result of *acquired immunity*.

Whooping-cough may occur *sporadically*, *endemically*, and *pandemically*, and not rarely children who leave their homes with whooping-cough in order to avail themselves of the favorable influence of change of air constitute the source of an epidemic in healthy places. In large cities epidemics of whooping-cough not rarely recur at fairly regular intervals, which generally vary from two to four years. Not rarely epidemics of whooping-cough immediately precede epidemics of measles, scarlet fever, Rötheln, or chicken-pox, or they may exist simultaneously, or the former may follow the latter. It may also happen that a child is at the same time affected with measles and whooping-cough, or with whooping-cough and chicken-pox, etc. Most epidemics of whooping-cough begin in the spring or the autumn.

Symptoms.—The *period of incubation* of whooping-cough is seven days. The local symptoms generally begin with the signs of bronchial catarrh (cough, râles), which extend, on an average, over two weeks, and constitute the **preliminary catarrhal stage** of whooping-cough. The true nature of the disorder is disclosed only when the catarrhal stage has passed over into the **spasmodic** or **convulsive** stage, which generally has an average duration of four weeks; occasionally, it is true, persisting for more than twice as long. In this stage the character of the cough is distinctive. The patients are generally at first conscious of a sense of tickling in the larynx or beneath the sternum, which they are incapable of resisting. Often the patients now grasp the nurse, or an object close at hand, for support during the paroxysm of cough. They also not rarely break out into tears. The actual coughing paroxysm begins with a deep, long-drawn crowing or whistling inspiration, which has been compared to the bray of an ass, whence the older name of "asses' cough" for whooping-cough was derived.

This deep inspiration is followed by a greater or lesser number of short expiratory efforts at cough, during which the child is wholly unable to breathe, and appears to be threatened with suffocation. Finally, another whistling, long-drawn inspiration takes place—the so-called *reprise*, or *whoop*—but which again is followed by numerous renewed efforts at coughing. Generally the paroxysm of cough does not cease until viscid mucus has been expelled from the larynx into the mouth, and which not rarely must be removed from the mouth of the little patients with the fingers.

During the paroxysm of cough signs of disturbances in the venous circulation make their appearance. The entire face and the skin of the neck acquire a bluish-red, cyanotic appearance, whence the name "*blue cough*," the jugular and the facial veins become distended into thick blue cords, the eyeballs protrude (exophthalmos), and the lips become greatly swollen, like sponges. Often the face, particularly the forehead, becomes covered with sweat. The duration of the individual paroxysm of cough varies between ten and thirty seconds, and but rarely persists for a longer time. The condition becomes especially distressing if one paroxysm of cough is immediately followed by a second and a third. In addition to the fear of suffocation the patient then suffers especially from pain in the head and vertigo, in consequence of intracranial circulatory disturbances.

The number of paroxysms of cough in the course of a day is extremely variable and determines the severity of the disease. In mild cases possibly from three to five seizures may occur, while in severe cases the number may be from sixty to one hundred, and even more, within twenty-four hours. After the termination of the paroxysm the patients usually recover with surprising rapidity, and soon resume their play with unaltered cheerfulness. The individual paroxysm of cough occurs either without exciting cause or in consequence of emotional disturbances, in laughing, or as a result of the entrance of foreign bodies into the larynx. Occasionally it can be induced by protrusion or by depression of the tongue. The paroxysms usually occur with greater frequency at night, because mucus readily accumulates in the larynx during sleep.

Examination of the bronchi and the lungs discloses either no alterations or signs generally of dry bronchial catarrh (sonorous and sibilant râles). On the other hand, distinctive alterations will be found in the **larynx** and the **trachea** on laryngoscopic examination. Laryngoscopic examination in children is, it is true, generally attended with great difficulty. The laryngeal mucous membrane, down to the true vocal bands, will be found vividly reddened —particularly the region between the arytenoid cartilages (interarytenoid space), which responds with especial readiness with cough to mechanical irritation—and also the posterior surface of the epiglottis. The redness extends down to the bifurcation of the trachea. Favorable conditions have permitted the observation that the beginning of a paroxysm of coughing is preceded by a collection of mucus in the interarytenoid space, and that at the conclusion of the paroxysm the mucus has disappeared. The application of a sound to the interarytenoid space or the posterior surface of the epiglottis will excite a paroxysm of coughing.

Whooping-cough ends with a *terminal catarrhal stage*—stage of decline or critical stage. The paroxysms of spasmodic cough become gradually less frequent and shorter, the expectoration more abundant and fluid, and finally, as at the beginning of the disease a simple bronchial catarrh exists, which also disappears in the course of about from two to four weeks, so that the average duration of an attack of whooping-cough is from eight to twelve weeks

The *general condition* suffers extremely little in many cases The patient is free from fever, remains out of bed, takes daily walks, and at most appears somewhat more pallid than usual.

Complications not rarely attend whooping-cough. In accordance with their causes they can be divided into two groups namely, those resulting from mechanical and those resulting from infectious influences. Among the *complications of mechanical origin* should be included all of those that result from the increased expiratory pressure induced by the spasmodic cough or from circulatory disturbances. *Rupture of blood-vessels* and *hemorrhages* beneath the skin and some of the mucous membranes occur frequently during the paroxysms of spasmodic cough. Subconjunctival hemorrhages especially, imparting a blood-red appearance to the white sclera, occur not rarely, and often permit the diagnosis of whooping-cough to be made at first glance. Occasionally a few drops of blood escape from the external auditory canal, in consequence of rupture of the tympanic membrane.

A frequent mechanical result of whooping-cough consists in *acute dilatation of the lungs*, which, if it do not subside, may develop into *alveolar pulmonary emphysema*. Rupture of the peripheral layers of the lung and of the pulmonary pleura may also occur, with the escape of air into the pleural cavity—*pneumothorax* In other instances rupture of the pulmonary tissue may give rise to *interstitial pulmonary emphysema*, which, however, can be recognized only if the air by passing along the peribronchial connective tissue to the anterior mediastinum, and higher up, gives rise to *subcutaneous emphysema* in the jugular fossa. Mechanical alterations in the *heart* also are often observed; dilatation of the right ventricle occurs particularly.

The *stomach* is involved with especial frequency, the violent succussion to which it is subjected through the movements of the diaphragm exciting *efforts at vomiting*. Some patients vomit for weeks after each paroxysm of cough, and in such abundance that their nutrition becomes impaired in an alarming degree. With regard to the *intestines*, it is often observed that during a paroxysm of cough the sphincter ani is not closed with sufficient firmness, so that gases and feces may escape involuntarily—*involuntary defecation. Prolapse of the rectum* and the development of *hernia* may also readily take place. Like the anal sphincter, the *sphincter of the bladder* also may be incompetent during a paroxysm of cough, and urine, in addition to feces, may escape involuntarily.

When whooping-cough occurs in a pregnant woman there will be danger of *abortion*, due probably alone to the violent succussion of the uterus. It should not be forgotten that *hemorrhage into the brain and into the cerebral meninges* is also possible, although it occurs but seldom. Especial consideration, because of diagnostic significance, is to be attached to shallow *ulcers upon and at the side of the frenum of the tongue*, which result from the impact of the inferior surface of the tongue against the lower teeth.

Among the *inflammatory complications bronchial catarrh* occupies first place on account of its frequency. The condition becomes serious when *bronchopneumonia* (*pertussis-pneumonia*) is superadded; this is generally revealed by elevation of temperature, and not rarely terminates fatally by asphyxia. Other inflammatory complications are less common.

Among the *sequelæ* of whooping-cough *pulmonary emphysema, hernia,* and *prolapse of the rectum* have already been mentioned. In some patients a *tendency to bronchial catarrh* and *undue sensitiveness of the respiratory organs* persist for a long time. Occasionally *pulmonary tuberculosis* develops. *Miliary tuberculosis* also, and especially tuberculous meningitis, are not unusual sequelæ, which occasionally appear only after the lapse of many years, and often originate from enlarged tuberculous-cheesy bronchial glands.

Diagnosis.—The recognition of whooping-cough is easy at the time of the convulsive stage, and generally a description of the paroxysms of cough by the members of the family is sufficient to render the diagnosis certain. In doubtful cases it should be recalled that frequently opening the mouth and depressing the tongue by means of the handle of a spoon, or a spatula, will excite a paroxysm of cough. The presence of external hemorrhages and of ulcers on the frenum of the tongue should also be looked for. In the catarrhal stage it is scarcely possible to differentiate pertussis from ordinary bronchial catarrh.

Prognosis.—Whooping-cough unattended with complications is a benign disease. If the number of paroxysms of cough is less than twenty during the day, the attack may be considered a mild one; while if the number is more than twenty the attack is a severe one, and the prognosis should be guarded. The disease often assumes a serious aspect if pneumonia occurs as a complication.

Anatomic Alterations.—No distinctive anatomic lesion ha as yet been found in cases of whooping-cough. Catarrhal inflammation of the upper air-passages and acute inflammatory swellin of the tracheobronchial lymphatic glands are frequently observed The lungs are often the seat of bronchopneumonic foci.

Treatment.—No *specific treatment* for whooping-cough i known. Resort must be had, therefore, to **symptomatic or expectant measures.**

It is true, a large number of specifics have been recommended, but non has, in my experience, sustained the claim made for it, not even *quinin.*

In uncomplicated cases quite as much can be accomplished b means of a suitable **mode of life** and **patience** as with drugs. Th room occupied by the patient should be as well ventilated as possible, as a crowded room with deficient ventilation favors th development of bronchial catarrh and bronchopneumonia. It i also well during the summer to spray the room thrice daily with 3 per cent. solution of carbolic acid, and during the winter to plac in the chimney or upon the stove a vessel containing water an 5 c.c. of creosote. On sunny and quiet days walks in the ope air are to be recommended, but care should be taken to avoid al association with healthy children, and the ejection of the sputu upon the street, in order to prevent infection of others. The die should be easily digestible, with a preference for milk.

When whooping-cough pursues a mild course drugs will b scarcely necessary. If, however, the paroxysms of cough occu frequently and in rapid succession, *narcotics* should be prescribed and among these especially belladonna-leaves, bitter-almond water and sodium bromid, are to be recommended. In the presence of extensive bronchial catarrh narcotics may be combined with *expectorants,* as, for instance:

℞ Infusion of belladonna-leaves, 0.5 : 80 (7½ grains : 2½ fluidounces)
Simple sirup, 20.0 (5 fluidrams).—M.
Dose: From 5 to 10 c.c. (teaspoonful to dessertspoonful) every two hours

℞ Infusion of ipecac-root, 0.3 : 80 (4½ grains : 2½ fluidounces)
Bitter-almond water, 5.0 (75 minims);
Simple sirup, 15.0 (½ fluidounce).—M.
Dose: From 5 to 10 c.c. (teaspoonful to dessertspoonful every two hours)

℞ Infusion of ipecac-root, 0.3 : 80 (4½ grains : 2½ fluidounces)
Sodium bromid, 5.0 (75 grains);
Simple sirup, 20.0 (5 fluidrams).—M.
Dose: From 5 to 10 c.c. (teaspoonful to dessertspoonful) every two hours

Attention should be given to the *prophylaxis. Association* of the healthy with the sick should be forbidden, and the *expectoration* should be received into a 5 per cent. solution of carbolic acid *Clothing, linen,* and the *sick-room* should be disinfected afte recovery has taken place.

INFLUENZA.

Etiology.—The exciting agent of influenza has been discovered, and was first found by Pfeiffer (in 1892) in the nasal and bronchial secretions alone as the **influenza-bacillus.**

Influenza-bacilli are slender, short bacilli, with rounded extremities that stain deeply with aniline colors, as, for instance, carbol-fuchsin, particularly at the extremities (Fig. 67), and can be readily grown in pure culture upon blood-agar, and successfully inoculated in monkeys.

Influenza exhibits the peculiarity of having disappeared from the earth for many years, so that at times physicians were familiar with the disease only from description; then the affection suddenly reappears, occasionally spreads within a short time over the entire earth in the form of a *pandemic*, then to disappear again for a longer or shorter period. Especially in the last decade pandemics and extensive epidemics have been observed, which have in part been characterized by their extreme malignancy. Influenza occurs at times in *endemic* distribution, being confined, for instance, to isolated barracks or prisons.

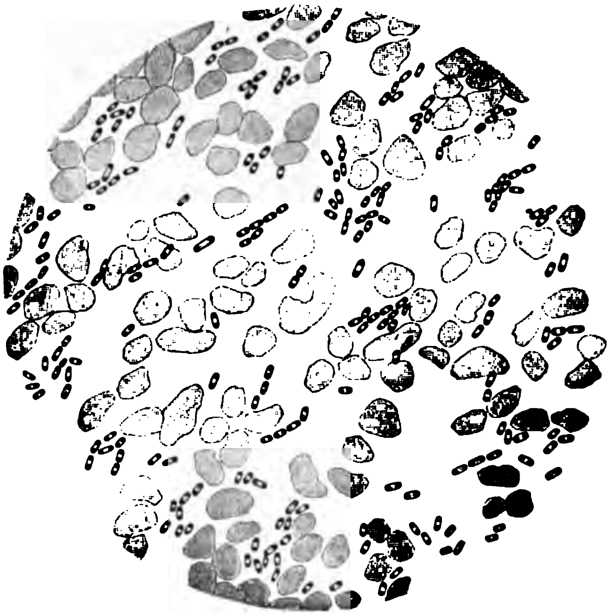


FIG. 67.—Influenza-bacilli from the sputum; carbol-fuchsin staining; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

The predisposition to influenza is quite widespread, and the period of incubation is probably short. Both of these circumstances render intelligible the fact that epidemics of influenza often develop with *explosive suddenness*, attacking a large number of persons almost simultaneously. Most epidemics occur in the autumn and the spring. Their duration varies between a few weeks and several months. Recovery from one attack of the disease does not confer protection from subsequent attacks. Some persons are attacked with influenza as often as they are exposed to the infective influences. Most frequently infection takes place through immediate *contact*. Infection through the *air* is conceivable only for a short distance, and not, as was formerly assumed, for hundreds of miles. Infection may take place, also, through the intermediation of a *third person* and of *inanimate objects*, although the influenza-bacillus is extremely sensitive to drying. In all probability, influenza-bacilli gain entrance into the body through the air-passages.

Symptoms and Diagnosis.—Nothing definite is known with regard to the *period of incubation* of influenza, although my own observations indicate that it is often only a few hours. *Prodromes* are wanting, or consist in general languor, dejection, a sense of oppression, pallor, and the like. The actual symptoms of the disease frequently set in with **chilliness**, less commonly with a single chill, which may be followed by elevation of temperature to 39° C. (102.2° F.) and above.

Three varieties of influenza have been distinguished, and they have been designated *bronchitic* (*thoracic*), *gastro-intestinal* (*abdominal*), and *nervous* (*cephalic*). Nevertheless, it should be borne in mind that the pure varieties of influenza are far less common than the mixed varieties. *Bronchitic influenza* is attended with symptoms of severe febrile catarrh of the upper air-passages. Nasal catarrh occurs, with prickling and burning in the nose, a sense of heat, a tendency to sneezing, and increased secretion. The voice becomes thick and hoarse in consequence of laryngeal catarrh, and severe cough and expectoration point to the presence of catarrh in the trachea and the bronchi. Over the lungs widespread sonorous, sibilant, and moist, but not ringing râles are generally heard. The patients complain of a feeling as if a foreign body were present beneath the sternum, sleep is disturbed by cough, and distressing pains occur in the thoracic muscles, largely in consequence of straining and stretching due to the efforts at cough.

Influenza can be distinguished with certainty from ordinary bronchial catarrh only by the presence of influenza-bacilli in the sputum, and this may frequently be demonstrated by means of a dry cover-slip preparation made from the sputum, and stained with carbol-fuchsin. Laryngotracheoscopic examination also discloses only diffuse swelling, redness, and increased secretion from the laryngeal and tracheal mucous membrane.

The inflammatory processes described generally invade adjacent mucous membranes. Through the lacrimal canal they extend to the **ocular conjunctiva,** where they give rise to redness and swelling, to increased secretion of tears, and to photophobia. The **mucous membrane of the frontal sinuses** becomes inflamed with especial frequency. The patients complain, in consequence, of intense pain on either side of the root of the nose, and pressure in this situation evokes tenderness. Pain in the superior maxilla, above the upper teeth, is suggestive of inflammation of the **mucous membrane lining the antrum of Highmore.** Frequently, roaring in the ears, impairment of hearing, and pulsatile pain in the ear are present—phenomena that are dependent upon extension of the inflammatory process along the Eustachian tube into the *tympanic cavity.* Suppuration and rupture of the pus externally through the tympanic membrane often occur. The **pharyngeal mucous membrane,** likewise, does not escape. A sense of burning and of tickling in the pharynx, and pain in swallowing, are indicative of inflammation of the pharynx, and inspection of the pharyngeal structures will disclose redness, swelling, and increased secretion.

The **general condition** suffers in marked degree in many cases. The patients are prostrated, feel excessively weak, as if they had been attacked by an especially grave disease, and not rarely become pallid with remarkable rapidity. With gradual subsidence of the fever and of the catarrhal symptoms recovery slowly sets in, and if the disease pursues an uncomplicated course it generally terminates in from seven to fourteen days, although occasionally it may occupy only from two to four days.

Complications, however, occur with extraordinary frequency, and especially the influenza-epidemics of the last decade have been justly notorious and feared on account of the frequent occurrence of serious complications. *Inflammatory complications* occur with especial frequency, in the causation of which particularly streptococci and pneumoniacocci take part, in addition to influenza-bacilli. Other complications, particularly certain nervous disturbances, are probably dependent upon the *action of bacterial poisons* (*toxins*) *of the influenza-bacilli.*

Among all the inflammatory complications *pneumonia* is most frequently observed, at times resembling bronchopneumonia, at other times rather fibrinous pneumonia, and occasionally being converted into *abscess and gangrene of the lung.* The latter condition I have observed to rupture into the pleura and to give rise to *putrid pyopneumothorax.* Also *pleuritis* and *pericarditis,* less commonly *myocarditis* and *endocarditis* occur. Occasionally marked gastro-enteritic symptoms are present, such as persistent and frequent *vomiting* and profuse *diarrhea.* At times the stools contain blood.

Often *nervous symptoms* are prominent—headache, vertigo, coma, delirium, even marked psychopathy, occasionally signs of encephalitis and cerebral abscess. Palpitation of the heart, and irregularity in the action of the heart, are also among the nervous complications. Troublesome neuralgia is especially disagreeable, at times being confined to a single nerve, or jumping from one nerve to another. Inflammatory processes in the peripheral nerves at times give rise to peripheral paralysis. The *organs of special sense* are often involved, especially inflammation of the eye and the ear occurring, and also paralysis of the ocular muscles. Arterial thrombi occasionally form rapidly in the *blood-vessels*, and they may cause gangrene of one or another extremity, and terminate fatally from septicemia.

Among the *sequelæ* protracted pallor, vertigo, irritability, and sleeplessness especially are to be mentioned. The complications that have been named may in part become sequelæ.

The *abdominal variety of influenza* is characterized especially by severe *vomiting* or profuse *diarrhea*, or both together. In women hemorrhages from the genitalia occur not rarely, and abortion often take place in pregnant women. In the *nervous variety of influenza* all of those disturbances may occur that have already been mentioned as complications of bronchitic influenza. A bacteriologic diagnosis is not possible during life. The recognition depends essentially upon whether gastro-enteritic or nervous symptoms are present during the prevalence of an epidemic of influenza without other discoverable cause.

Prognosis.—Uncomplicated influenza is readily recovered from by young and robust individuals, but is a serious disease for the *debilitated*, and especially for the *aged*, in whom death readily results from exhaustion. The prognosis depends essentially upon the nature of the *complications*, and, as these vary with the *character of the epidemic*, the latter will also determine the gravity of the prognosis.

Anatomic Alterations.—Distinctive anatomic lesions are not known. The **mucous membrane of the air-passages** is the seat of redness and swelling. The **spleen** is generally soft and diffluent (infection-spleen). The **kidneys** are hyperemic. Influenza-bacilli have been demonstrated in the **brain** in cases of cerebral influenza. They have been found, also, in the **intestine**, in the spaces of the follicles of Lieberkühn, as well as between the epithelial cells and in the basement-membrane. The remaining alterations in the intestines are suggestive of cholera.

Treatment.—No *specific treatment* for influenza is known. Generally the patients are so distressed by individual symptoms that **symptomatic treatment** must be instituted, and expectant treatment cannot be followed. In cases of bronchitic influenza the air of the sick-room should be kept moist by spraying a 3 per cent. solution of carbolic acid thrice daily; or, during the winter, by

keeping in the chimney or upon the stove a vessel containing water to which 5 c.c. of creosote have been added. As a beverage *warm infusions* may be recommended, as, for instance, pectoral species—one tablespoonful to a cup of hot water several times daily. Severe irritative cough should be relieved by means of mild *narcotics*, as, for instance :

℞ Powdered ipecac and opium,	0.3 (4½ grains).
Dose : thrice daily.	

Or,

℞ Bitter-almond water,	10.0 (2½ fluidrams);
Morphin hydrochlorate,	0.01 (⅙ grain).—M.
Dose : 10 drops thrice daily.	

Resort to *expectorants* will also have to be made frequently. Subcutaneous injections of pilocarpin hydrochlorate (0.3 : 10.0—4½ grains : 2½ fluidrams ; half a syringeful—8 minims—subcutaneously) have been lauded. For the relief of neuralgia and other nervous disturbances *sodium salicylate*, *phenacetin*, *antipyrin* (1.0—15 grains—thrice daily) especially are to be recommended. Also, in the presence of slight fever rest in bed is to be advised. The patient should avoid going out into the open air too early, as relapses readily occur under such circumstances. As a *prophylactic* measure isolation of the patient should, so far as possible, be secured and the sputum rendered innocuous by its reception into a 5 per cent. solution of carbolic acid.

V. INFECTIOUS DISEASES ATTENDED WITH LOCAL ALTERATIONS IN THE DIGESTIVE ORGANS.

EPIDEMIC PAROTITIS.

Etiology.—Epidemic parotitis is known also as *mumps*, and is an infectious disease that occurs in epidemic distribution most frequently in the autumn or the spring. The *infectious agent* has not yet been discovered. In all probability it gains entrance through the mouth into the duct of Stenon, and from here into the parotid gland. The small caliber of the duct of Stenon in infants has been assumed to be the cause for the infrequency with which the disease appears in them.

Infection generally occurs through *personal association*. It has been observed in educational institutions that almost all of the pupils are attacked by mumps if at the close of the vacation one of the pupils returns from home with parotitis. In children schools and public playgrounds are the principal channels through which infection takes place, and not rarely epidemics are confined to a single school.

Symptoms.—The *period of incubation for epidemic parotitis* may be as long as twenty-one days, although frequently it is less. *Prodromes* often are wholly wanting. Occasionally the disease sets in with **chilliness and slight elevation of temperature.** In other instances, however, the general condition remains unaffected and **local changes in the parotid gland** appear earliest. These begin at first in one gland, according to some most frequently in the left, and then in the course of one or two days they make their appearance also in the other gland. In the latter, however, they may be so ill developed as to be overlooked altogether on superficial examination. The first symptoms generally consist in a sense of tension, or even in slight pain in the affected parotid gland. The involved region becomes enlarged almost to the size of a fist, and above, and feels hard and often also somewhat hot. The overlying skin is tense, glistening, and smooth, and more often pale than red in consequence of compression of the vessels.

The swelling, in correspondence with the situation of the parotid gland, occupies the region between the mastoid process, the malar arch, and the angle of the lower jaw; but it extends also, particularly in the submaxillary region into the neighborhood. The patient is in great degree embarrassed in depressing the lower jaw, and if marked enlargement of the parotid gland is present upon both sides he will scarcely be able to separate the teeth. He speaks through his teeth, and is only with difficulty capable of taking solid and liquid food.

In consequence of imperfect cleansing of the mouth the **breath** often acquires a disagreeable [illegible] the movements of the head are interfered with [illegible] the **head** is generally [illegible] toward the affected [illegible] relax the skin of the face [illegible] The **secretion of saliva** is [illegible] the **reaction of the saliva** [illegible] face is greatly distorted [illegible] The **expression** becomes [illegible] of derision for the friends. [illegible] teen days the swelling [illegible] becomes more and more [illegible] desquamation of the skin [illegible]

Among the anomalies [illegible] indicated in which not the [illegible] the submaxillary gland [illegible] may also happen that [illegible] occurs while the salivary glands [illegible] other cases it is true [illegible] parotitis but must be considered [illegible]

Among the *complications inflammation of the testicle—orchitis*—is the most frequent and the most important, although this occurs only in virile males. It is almost always unilateral, and is attended with a sense of heaviness, pain, and swelling in the affected organ. At times the swelling of the parotid has been observed to subside, while orchitis developed, and to become more active after the orchitis had gotten well. Occasionally orchitis is followed by *atrophy of the testicle,* in consequence of which, if it be bilateral, *sterility* in the male may result. In women swelling and tenderness of the breasts, or of the ovaries, and the formation of a hematoma of the labia have been observed. *Suppuration* occurs but rarely in the diseased parotid gland, and it may be attended with rupture of the pus externally or with burrowing abscesses, with erosion of the large blood-vessels of the neck, even with burrowing and secondary suppuration in the pleural or the pericardial cavity, or in the mediastinum, together with its various dangers, or with general septicemia. At times *disease of the ear* occurs, particularly of the labyrinth, which may induce vertigo, vomiting, impairment of hearing, or deafness.

Of the *sequelæ of epidemic parotitis* some have already been mentioned as occurring as complications. Occasionally the inflamed parotid gland is not wholly restored to its normal size, but remains enlarged and indurated. Either *ptyalism* or *diminution in the secretion of saliva* may also occur. Pressure upon the expansion of the facial nerve is occasionally attended with *peripheral facial palsy.*

Diagnosis.—The recognition of epidemic parotitis is easy on account of the distinctive local alterations. It is distinguished from **secondary** and **metastatic parotitis** occurring in the sequence of a number of infectious diseases, in the first place, by the circumstance that it occurs as an independent disease, and, further, that it but rarely gives rise to suppuration.

Prognosis.—Epidemic parotitis is a benign disorder, which scarcely ever terminates fatally.

Anatomic Alterations.—Little of a definite nature is known with regard to the anatomic alterations, as few opportunities are afforded for study. It is assumed that catarrhal inflammation of the excretory ducts and the acini of the gland arising through the salivary ducts is present.

Treatment.—As no *specific remedy* is known, the treatment is restricted to **symptomatic,** and especially **antiphlogistic measures.** The swollen portion of the face is rubbed with warm *olive-oil,* and is covered with *absorbent cotton.* The mouth is rinsed after each meal with a *solution of aluminum acetate* (1.0 : 200). The *diet* should be liquid, with a preference for milk. Even if the disease pursue an afebrile course the patient will do well at least to remain in his room. If orchitis develop, the inflamed testicle should be elevated upon a pillow or upon a supporting towel, and enveloped in compresses that have been saturated with lead-water.

ACUTE PHLEGMONOUS PHARYNGITIS.

Etiology.—But a few instances of this disorder have been recorded. It occurs as an independent infectious disease, but the causative agent is as yet unknown.

The **anatomic alterations** consist in purulent infiltration of the deeper layers of the pharyngeal mucous membrane extending downward to the laryngeal mucous membrane. The adjacent lymphatic glands are hyperemic and enlarged. In addition, acute splenic enlargement is present.

Symptoms.—The patients complain of **difficulty in swallowing** and **pain in the pharynx**, and on inspection the **pharyngeal mucous membrane** will be found to be greatly reddened and thickened. Often the **cervical connective tissue** also is inflamed, and consequently reddened and infiltrated. Involvement of the **larynx** in the inflammatory process will be indicated by hoarseness and pain over the larynx. Occasionally acute inflammatory swelling of the laryngeal mucous membrane, particularly that of the false vocal bands and the aryepiglottic folds, develops within a short time, and the patients are seized with a marked degree of shortness of breath, and unless speedy relief be afforded death may result from suffocation. **Diarrhea** not rarely occurs. The **spleen** is enlarged. **Fever** and **delirium** are generally present.

Diagnosis.—The disorder may be most readily confounded with **erysipelas of the pharyngeal mucous membrane,** although streptococci have hitherto not been found in the pus from pharyngeal phlegmons.

The **prognosis** is grave, as death may result from septicemia or acute edema of the glottis.

The **treatment** consists in the application of an **ice-collar,** in sucking of **bits of ice,** in **incision** of such collections of pus as may be present, and, in the presence of edema of the glottis, in **intubation** or **tracheotomy.**

TYPHOID FEVER.

Etiology.—The exciting agents of typhoid fever are in all probability the **typhoid-bacilli** carefully studied by Eberth and Gaffky. They are found constantly and exclusively in cases of typhoid fever. They have been grown in pure culture, the growth especially upon discs of sterilized potato exhibiting a characteristic appearance, although the micro-organisms have hitherto not been successfully inoculated upon animals, obviously because these are not attacked by typhoid fever. Although animals die after inoculation with cultures of typhoid-bacilli, death occurs as a result of poisoning (intoxication) and not of infection, for their organs do not exhibit any anatomic alterations of typhoid fever.

The *typhoid-bacilli* are small rods with rounded extremities (Fig. 68), which do not stain with especial readiness with aniline dyes, but best with Löffler's methylene-blue, and which yield up their color when stained by the method of Gram. They often lie together in pairs and in considerable number, and frequently exhibit an unstained circular area at one pole that is considered a spore. Should one bacillus be continuous with another, the spores will be situated in the adjacent poles. In fresh preparations, and in the hanging-drop, typhoid-bacilli exhibit active, *independent movement*, which is effected by flagella, of which six, and even more, arise from the sides and extremities of the body. In order to demonstrate the flagella special staining-methods are necessary. If blood-serum from a case of typhoid fever be added to a bouillon-culture of virulent typhoid-bacilli, loss of motility occurs within a short time, with agglutination of the previously disseminated and actively swarming typhoid-bacilli. This change can be recognized macroscopically from the fact that the previously homogeneously turbid bouillon becomes clear, the cloudy and flocculent turbidity due to the agglutinated typhoid-bacilli being thrown down as a precipitate. Microscopic examination discloses immobile collections of bacteria. This procedure constitutes the *blood-serum reaction* of Widal, and it can be employed for the purpose of determining that doubtful bacilli are typhoid-bacilli if the blood-serum is derived from a case of typhoid fever, or that a doubtful disease is certainly typhoid fever if one can be sure that he is dealing with typhoid-bacilli. The latter test is of especial importance to the practitioner. Although the blood-serum of healthy individuals likewise possesses feeble agglutinating and inhibiting power, it manifests this property only in a dilution of about one to ten of bouillon-culture of typhoid-bacilli. The blood-serum from a case of typhoid fever induces the effects described in dilutions of from one to twenty to one to a hundred and more.

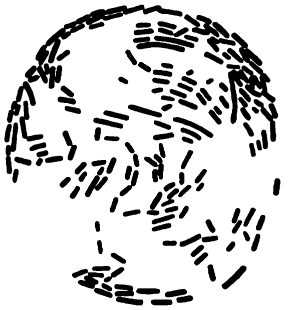


FIG. 68.—Typhoid-bacilli: smear-preparation from a pure culture prepared from the stools from a case of typhoid fever in the Zurich clinic; magnified 1250 times.

Typhoid-bacilli are given off from the body of typhoid patients especially with the *feces* and the *urine*, and it is through both of these sources that further infection is brought about. Infection occurs with especial frequency through the *use of water* and of articles of food to which water has been added, as, for instance, milk, if urine or intestinal discharges from a case of typhoid fever have been added to the water. Such contamination may take place readily if privy-wells are not cemented and water-tight, and if situated in the vicinity of springs into which fluid contents of cesspools, together with typhoid-bacilli, may leak through the intervening soil. Also, if the intestinal discharges and the urine from cases of typhoid fever are thrown, without previous disinfection, into brooks and streams whose water is used for drinking-purposes at a lower level, typhoid-bacilli may be disseminated in a most luxuriant and successful manner. Washing in brooks of the linen from cases of typhoid fever may also be a source of danger, as such clothing is usually contaminated with fecal matter and urine. Under such circumstances an epidemic of typhoid fever may develop within a few days, and may extend with approximate uniformity throughout an entire community, if it use the infected water. The influence of water in the dissemination of typhoid fever has been clearly demonstrated especially when a community has been provided with two sources of supply, of which only one was contaminated with typhoid-bacilli, and only those, or, at any rate, principally those who used the water from the infected source being attacked with typhoid fever. It is thus evident how important it is for every community to provide itself with a thoroughly protected and closed water-supply and a water-tight canal-system of drainage. In the selection of a source of water-supply it should not be overlooked that this be obtained from remote and unbuilt-up localities and from the deeper layers of the earth, in order that it may not be contaminated with typhoid-bacilli in manuring fields and meadows. It is also important that water for general supply be freed as thoroughly as possible from lower forms of organic life by means of sand-filtration.

Typhoid-bacilli have in a number of instances been demonstrated in water after the occurrence of epidemics of typhoid fever, although such examination is attended with great difficulty, as there are more than thirty varieties of bacteria that closely resemble the typhoid-bacillus.

Nurses and *laundresses* are exposed to the danger of swallowing typhoid-bacilli directly if, in the care of the typhoid patient or in washing the linen, small particles of fecal matter become attached to the hands and the fingers and food be subsequently taken without previous disinfection of the hands. Infection through *inhalation of typhoid-bacilli* occurs but rarely. I have observed such an occurrence in an attendant at my clinic, who during the summer had beaten mattresses that had been used several months previously by patients suffering from typhoid fever. The *wearing of undisinfected clothing from typhoid patients* also has occasionally given rise to an outbreak of typhoid fever. Such a mode of infection has been observed in soldiers, and experience has taught, in this connection, that typhoid-bacilli may retain their vitality in clothing for several years.

The possible modes of infection are by no means exhausted with those mentioned. Among other things it should be recalled that the use of *thermometers, of syringes for enemata*, and *of bed-pans*, first in cases of typhoid fever, and then by another patient without previous adequate disinfection of the utensils, may result in immediate conveyance of typhoid fever. Occasionally epidemics of typhoid fever occur without appreciable cause. It should be recalled, however, that some patients appear perfectly healthy and pursue their avocations without interruption in spite of the existence of typhoid fever, although the intestinal discharges may be a source of infection.

Typhoid fever has been definitely known only since the middle of the nineteenth century. Until about within the last decade it was an exceedingly common disorder in some cities, which, as a result, acquired an evil and not undeserved reputation. Since, however, a beginning has been made in the provision of a proper water-supply and drainage, typhoid fever has become a rare disease also in notoriously typhoid cities.

Typhoid fever now and then occurs *sporadically*, and then attacks especially *strangers* who have acquired the disease elsewhere. Sporadic cases of the disease occasionally furnish the source for an *epidemic of typhoid fever*. Epidemics of typhoid fever are at times confined to certain houses with many inmates, as, for instance, barracks.

The occurrence of epidemics of typhoid fever does not depend upon the *season of the year*. *Age*, likewise, is without influence, although most cases of typhoid fever occur between the twentieth and the fortieth year of life.

In *pregnant women* typhoid-bacilli may gain entrance into the blood and viscera of the fetus through the placental circulation. Occasionally the anatomic alterations of typhoid fever have developed in the fetus.

Recovery from one attack of typhoid fever confers protection, as a rule, from subsequent attacks; but rarely some individuals may be attacked as many as four times by typhoid fever. Attention has been directed to the fact that the members of some families are attacked by typhoid fever with unusual frequency, so that a *familial predisposition* has been assumed to exist. It has also been observed in numerous instances that if during an epidemic of typhoid fever several members of a family are attacked the disease often pursues a remarkably similar course.

Occasionally *typhoid fever occurs in association with some other infectious disease* in the same person, as, for instance, with measles, scarlet fever, erysipelas, herpes zoster, tuberculosis, syphilis, etc.

Symptoms.—The *period of incubation of typhoid fever* is from fourteen to twenty-one days. *Prodromes* are either wanting, or are manifested by chilliness, slight and irregular febrile movement, general malaise, anorexia, disturbed sleep, and neuralgic symptoms, especially neuralgia of the occipital nerves, or of some branches of the trigeminus. Complaint is often made of pain in the muscles of the thighs. The distinctive morbid manifestations often set in with repeated chilliness rather than with a single chill. The fever becomes continued, and gradually the remaining symptoms of typhoid fever appear, among which the typhoid tongue, the roseolæ, the abdominal distention, the enlargement of the spleen, the typhoid stools, and the blood-serum reaction may be mentioned.

The **course of the fever** is peculiar and of diagnostic importance. In the first week of the disease, during which medullary swelling of the lymph-follicles of the intestinal mucous membrane occurs, the temperature slowly rises—step-like. In the second week of the disease, when necrosis and exfoliation of the intestinal lymph-follicles, the seat of medullary inflammation, take place, the temperature reaches its highest level (from 39° to 41° C.—102.2°–105.8° F.), and it then remains continued. In the third week of the disease, in the stage of ulceration of the intestine, greater daily fluctuations in the temperature take place, which usually become even more marked in the fourth week, in the stage of cicatrization of the ulcers, so that a *hectic stage* has been spoken of, and with a gradual decline of the fever the temperature reaches the normal level. The fever of typhoid, therefore, does not terminate by crisis, but by lysis.

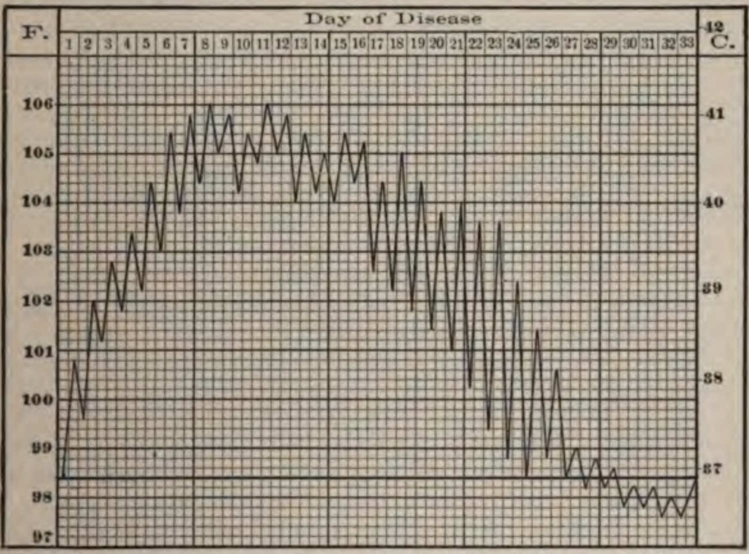


FIG. 69.—Temperature-curve from a case of enteric fever (Salinger and Kalteyer).

It is worthy of consideration that the pulse-frequency of typhoid fever is generally less than would correspond with the elevation of bodily temperature, so that with a temperature of more than 39° C. (102.2° F.) the pulse is frequently below 100. The pulse also is frequently characterized by marked dicrotism, particularly in the second half of the disease.

In addition, the usual febrile symptoms are present, viz., increased thirst, impaired appetite, increased frequency of respiration, and diminished diuresis. The skin, in the first half of the disease, is dry and hot, while in the fourth week often profuse sweating is present, and upon which, probably, also the marked fluctuations in temperature of the hectic stage are dependent. The skin is then, particularly in the morning, covered with numerous crystalline miliary vesicles, which are appreciable especially upon the lateral aspects of the abdominal wall as innumerable small elevations when the hand is passed over the surface.

In the first day of the disease the tongue is generally sticky and moist, and presents upon its dorsum up to the tip a grayish, grayish-yellow, or grayish-brown coating, consisting of desquamated squamous epithelial cells, round cells, remains of food, and bacteria. The margins of the tongue often show the marks of the teeth. Toward the end of the first week of the disease the tongue becomes dry and unusually red, often brick-red. In addition, the coating of the tongue is exfoliated at the tip, and thus a triangular, completely clean, and vividly red area comes into view on the anterior portion of the tongue, to which the designation *typhoid triangle* has been applied. The exfoliation of the coating of the tongue gradually extends over the dorsum of the entire organ.

Toward the end of the first week of the disease circular red spots, averaging the size of a pea and generally slightly raised, appear upon the skin—**roseolæ**—which fade upon pressure with the finger, and, accordingly, are dependent upon circumscribed hyperemia of the skin. The first spots usually appear at the junction of the chest and the abdomen, but they occur also upon the remaining portions of the abdomen and the chest, upon the surface of the entire back, occasionally upon the neck, and even upon the extremities, but never upon the face. They are extremely variable in number, which occasionally bears some relation to the entire character of the epidemic. Roseolæ constitute an almost constant manifestation of typhoid fever, and are, therefore, of great diagnostic importance. No prognostic significance can be attached to their number. The view that they may be due to embolism of cutaneous vessels with typhoid-bacilli, merely because typhoid-bacilli have been found in the blood contained within them, has not been sustained. The individual roseola persists for from three to seven days. Occasionally the skin undergoes slight desquamation after its disappearance. At times fresh roseolæ appear after defervescence and during convalescence.

The **abdomen** of a typhoid patient is distended in greater or lesser degree, as the intestine is distended in consequence of abundant development of gas. Especial consideration should be given to the *ileocecal region*. This is often extremely tender on pressure, and on palpation frequently yields *intestinal gurgling* (*ileocecal* *murmur*), which is due to pressure upon a loop of intestine filled with fluid contents and bubbles of gas. Often the right ileocecal region is dull on percussion as compared with the left iliac fossa.

The **spleen** will be found to be enlarged after the second half of the first week of the disease. Of especial diagnostic significance is the fact that the spleen can be felt when the body is placed in the right oblique position, as enlargement of the spleen as demonstrated by percussion only is not trustworthy, and may be readily confounded with especial readiness with transitory excessive distention of the splenic flexure of the colon. With the onset of recovery the enlargement of the spleen also slowly subsides. Should splenic enlargement persist during convalescence, a relapse may be suspected.

The **bowels** are generally constipated during the first days of the disease, although diarrhea generally sets in toward the end of the first week, so that, from this time onward, from two to six thin stools are voided daily. The typhoid stool is yellowish-green in color and alkaline in reaction, and on standing it deposits a light-yellow friable sediment. Its appearance has not inaptly been compared to that of pea-soup, and it has therefore been designated *pea-soup stool.*

Microscopic examination of the typhoid stool discloses the presence of remains of food, round cells, isolated red blood-corpuscles, innumerable bacteria, and crystals of ammonio-magnesium phosphate (coffin-lid variety). In the second week of the disease typhoid-bacilli can be obtained from the stools, although their growth in pure culture requires the services of a trained observer. Typhoid-bacilli may be confounded with especial readiness with the Bacterium coli, which is encountered in the intestinal discharges of perfectly healthy individuals. The principal distinction between these two bacteria consists in the circumstance that typhoid-bacilli, in contradistinction from colon-bacilli, do not cause fermentation of glucose-agar.

The presence of the **blood-serum reaction** previously mentioned is especially important in the diagnosis of typhoid fever, and in doubtful cases is decisive, but it is not to be expected before the termination of the first week of the disease.

Should no complication arise, recovery will generally be so far advanced after the lapse of six weeks that the patient may with some care resume his customary pursuits. Occasionally *relapses* occur, the causes for which consist in getting up too early, errors in diet, and emotional disturbances, but for which often no cause can be determined. Relapses generally pursue a favorable course. In rare instances several relapses may occur.

Among the *anomalies of typhoid fever abortive typhoid fever* may first be mentioned. Obviously, under such conditions the changes in the lymph-follicles of the intestine do not advance beyond medullary swelling, after which the anatomic process in the intestine subsides and undergoes involution. In conformity with this fact the fever persists for only one or two weeks, and recovery then sets in. In cases of *mild typhoid fever* the symptoms of the febrile general infection are but little marked. In rare cases the disease pursues an almost wholly afebrile course—*afebrile typhoid fever*—and occasions so little discomfort that the patient is capable of continuing at work without interruption—*ambulatory typhoid fever*. Especially patients of the latter kind constitute a source of great danger for the dissemination of the disease, as they are wholly unconscious of its presence and associate with others without restriction as do healthy persons. In addition, no source of infection can be discovered when further dissemination of the disease takes place.

Occasionally typhoid fever is concealed behind the clinical picture of fibrinous pneumonia, acute nephritis, or meningitis, and only gradually do the symptoms of typhoid fever come more prominently into view. Under such conditions the designation *pneumotyphoid*, *renotyphoid*, or *meningotyphoid* has been employed. The view that under such circumstances typhoid-bacilli have first lodged in the lungs, the kidneys, or the cerebral meninges, and then in the intestine, has not been shown to be correct.

Complications of typhoid fever depend either upon the intensity of the general infection and the duration of the disease, or upon ulcerative processes in the intestinal wall, or they may result from the dissemination of typhoid-bacilli or other pyogenic agents, particularly streptococci, in other organs.

Profound general infection is not rarely disclosed by *derangement of cerebral activity*, whence the name *nervous fever* instead of typhoid fever. The patients become comatose and restless, and delirium sets in. Occasionally they become so excited that they shout aloud and throw themselves about, become violent, spit upon those about them, jump out of bed, and, if not carefully watched, escape from the sick-room and throw themselves from the window to the ground. The ingenuity displayed by the patient in eluding observation is not rarely remarkable. Under such conditions the designation *versatile nervous fever* was formerly employed. The opposite condition consists in *stupid nervous fever*. In the latter condition also the patient is comatose, but his attitude is a quiet one. At most he mutters unintelligible words, but is otherwise apathetic, not asking for food or drink, failing to void urine unless directed, and permitting the bladder to become over-distended, and lying for hours in the most uncomfortable positions. Now and then he moves some fingers spasmodically, and the related tendons upon the dorsum of the hand and the forearms become prominent—so-called *subsultus tendinum*—or the patient tugs at the bed-clothing with his fingers as if unravelling wool—
Patients of this sort also require especially careful
They should be offered fluid to drink at

regular intervals, and the lips and the tongue should be moistened, and the latter also rubbed with oil morning and evening, in order to prevent excessive drying, cracking and bleeding, and desiccation of the blood into blackish-red deposits—fuligo. The patient should be directed to change his position every two hours, as otherwise gangrene of the skin from pressure may readily result. Regular evacuation of the bladder should also be provided for in order to prevent uremic intoxication, etc.

Profound and long-continued general infection is occasionally followed by an alarming condition of *myocardial weakness*. The myocardium becomes dilated, particularly the right ventricle, the apex-beat becomes enfeebled, even impalpable, the heart-sounds become scarcely audible, especially the first sound, the pulse becomes small and unusually frequent, also irregular, and if speedy relief be not afforded death results from cardiac paralysis.

Marantic venous thrombosis also is worthy of mention. It is most frequently encountered in the left femoral vein. It is attended with pain, a sense of coldness and weakness in the affected member, and can be readily recognized from the presence of cutaneous edema in the affected part alone, from the dilatation and tortuosity of the cutaneous veins and the lower temperature of the skin. It is well to refrain from any attempt to determine by palpation whether the venous thrombus can be recognized as a firm plug to the inner side of the pulsating femoral artery, because such manipulation may readily result in detachment of a fragment of the thrombus, with its entrance through the inferior vena cava into the right heart, and thence into the pulmonary artery, where multiple emboli may form, and if the main trunk of a pulmonary artery be obstructed sudden death may result.

Bilateral thrombosis of the femoral vein occurs but rarely, a thrombus in one femoral vein extending through the interposition of the vena cava into the distribution of the opposite femoral vein, or thrombosis developing in both femoral veins independently.

Marantic venous thrombosis is, as a rule, unattended with serious danger to life, but it retards recovery, as, occasionally, many weeks may elapse before the disturbed venous circulation is restored.

Marantic thrombosis of the cerebral arteries, particularly of the left Sylvian artery, with right hemiplegia and aphasia, is rather a sequel than a complication, and occurs but rarely.

Among the complications dependent upon the inflammatory and ulcerative processes in the intestinal mucous membrane, *excessive diarrhea* may first be mentioned. Thus, it occasionally happens in some cases of typhoid fever that more than from ten to fifteen bowel-movements occur within twenty-four hours. This circumstance is attended with the danger that death may result from exhaustion. Persistent *constipation* should be included among the intestinal complications, and this is observed not without disquietude, because it may readily cause premature exfoliation of the typhoid crusts and intestinal hemorrhage, or even perforation of the bowel. Typhoid fever is sometimes attended with marked *intestinal meteorism.* The intestines, over-distended with gas, fill the abdomen almost to the point of rupture, and the diaphragm, the lungs, and the heart are pushed upward to such a degree that death from suffocation is threatened, and at times actually occurs.

Intestinal hemorrhage does not generally occur before the third week of the disease, as it results from detachment of the typhoid eschar before the subjacent blood-vessels are closed with sufficient firmness by thrombi. Occasionally, its occurrence is preceded by the appearance of streaks of blood in the intestinal discharges, or of small amounts of blood recognizable only on microscopic examination; often, however, the accident occurs unexpectedly. The stool acquires a blackish-red, bloody appearance, and is admixed with other intestinal contents, or it is almost pure blood. In the latter event it frequently consists of masses of blood-clot. The amount of blood may reach one or two liters. If the loss of blood be considerable, the patient may present a death-like pallor, the bodily temperature declines, occasionally below the normal, consciousness, if previously obtunded, clears up, the skin feels cool, and dulness on percussion often appears in the right iliac fossa. From the characteristic appearance of the patient experienced clinicians will have suspected the occurrence of intestinal hemorrhage even before the bloody stool has shown itself. If the loss of blood be large, the patient occasionally dies without the discharge of a bloody stool. In some cases hemorrhage from the bowel is terminated with a single bloody stool, while in others the bleeding persists for from one to three days; rarely, considerably longer. It may also happen that it is for the time being controlled, but that in the course of a few days it is repeated once or even several times. Under all circumstances intestinal hemorrhage complicating typhoid fever is a dangerous occurrence.

Ulceration that has advanced deeply and has almost reached the serous layer of the intestine readily gives rise to *perforation of the bowel* and *perforative peritonitis.* The latter may be sacculated or free, and in the first event not rarely pursues so insidious a course that it may wholly escape recognition during life in spite of the most careful observation and examination. In some instances the intestinal wall escapes perforation, but circumscribed, encapsulated, or diffuse *free peritonitis* develops, which, like perforative peritonitis, greatly aggravates the prognosis under all circumstances.

Whether *inflammatory complications* are induced by conveyed typhoid-bacilli, streptococci, or other phlogogenic bacteria can be decided only by bacteriologic examination of the inflammatory products. It is noteworthy that typhoid-bacilli may retain their vitality in collections of pus for many years. Any viscus may be the seat of inflammatory complications in the course of typhoid fever. In the following a few instances may be cited.

Among the lesions of the respiratory organs *ulceration of the laryngeal mucous membrane* may be mentioned, occurring with especial frequency upon the free margin of the epiglottis, the vocal-band processes of the arytenoid cartilages, and the adjacent portions of the true vocal bands. In some cases typhoid-bacilli have been found on the floor of the ulcer. At times the ulceration gives rise to *edema of the glottis* or to *perichondritis of the larynx*, both of which conditions are of serious significance.

Bronchial catarrh is a frequent complication of typhoid fever. Occasionally, foci of *bronchopneumonia* or of *lobar pneumonia* develop in the lung. Frequently these result from the entrance of saliva or of particles of food into the lower air-passages, and they are, therefore, forms of aspiration-pneumonia or of foreign-body pneumonia. Not rarely aspiration-pneumonia becomes converted into *abscess* and *gangrene of the lung*. Persistent occupancy of the same position favors the development of *hypostasis of the lung*, and in turn of *hypostatic pneumonia*. *Pleurisy* is a well-known but not exceedingly common complication of typhoid fever. *Pericarditis, verrucose* and *ulcerative endocarditis* and *myocarditis* are among the less common complications.

Abscess of the liver, suppurative or gangrenous lesions of the gall-bladder, and *abscess of the spleen* occur but rarely in cases of typhoid fever. *Acute nephritis*, however, is encountered more frequently. *Angina* is common, at times catarrhal, at other times lacunar, and at still other times necrotic. Occasionally grayish-white deposits upon the tonsils also are found, consisting of desquamated epithelial cells and bacteria—cachectic angina. *Meningitis, encephalitis, myelitis*, and *neuritis* are not infrequently observed. Occasionally *periostitis* develops. In some cases of typhoid fever I have observed *septicopyemia* to occur and terminate fatally.

A number of *sequelæ of typhoid fever* result immediately from the complications, while others must be conceded a certain self-dependence and independence. Occasionally *enfeeblement of cerebral activity* persists for an exceedingly long time. The patient is forgetful, tires readily on mental exertion, is disinclined to intellectual activity, and only gradually reacquires his former vigor of mind. It occasionally occurs also that a *fixed idea* becomes established, which at times disappears only after the lapse of several weeks. In some patients *edema* and *hemorrhages into the skin* occur [illegible] the first efforts to get up. Occasionally *enlargement* *of the spleen* remains permanently. *Derangement of intestinal activity* also occurs, at times a tendency to diarrhea, at other times to constipation. A manifestation of no importance is *loss of hair* in consequence of nutritive disturbances in the hair-papillæ. The hair, however, almost always grows again perfectly.

Diagnosis.—The diagnosis of typhoid fever has been rendered most certain within the last few years by means of the *blood-serum reaction of Widal*, so that confusion with **miliary tuberculosis, ulcerative endocarditis,** and **septicopyemia** can generally be avoided. Nevertheless, the diagnostic significance of the blood-serum reaction is surrounded with certain limitations, for, in the first place, it does not appear before the beginning of the second week of an attack of typhoid fever, and, besides, it may occasionally persist for more than ten years in the blood of individuals who have at some time in their life recovered from an attack of typhoid fever. Practitioners who have neither sufficient time nor experience and the necessary apparatus for making bacteriologic examinations will do well to avail themselves of the services of a bacteriologic institute, or of a clinic, for which purpose it will be sufficient to take up with bibulous paper a few drops of blood from the patient, and to transmit this paper. The desiccated blood will then be dissolved in water, with which the reaction will be made.

In the majority of cases the clinical symptoms (course of the fever, typhoid tongue, typhoid abdomen, roseolæ, enlargement of the spleen, pain, dulness and intestinal gurgling in the ileocecal region, typhoid stool) further will suffice for the establishment of the diagnosis with certainty. The great difficulty of establishing the diagnosis by the demonstration of typhoid-bacilli in the blood, the urine, or the stools has already been pointed out. Puncture of the spleen and the demonstration of typhoid-bacilli in the splenic juice are not measures to be recommended.

Prognosis.—The prognosis of typhoid fever is not unfavorable so long as serious complications are not present, and the patient is a young, robust individual. When certain complications occur, particularly intestinal perforation and perforative peritonitis, recovery can scarcely be expected.

Anatomic Alterations.—The *characteristic lesions of typhoid fever* consist in the changes in the lymph-follicles of the intestine, in the mesenteric glands, and in the spleen. The *intestinal lymph-follicles* begin to enlarge, and in the course of the first week of the disease enter upon a condition of **medullary swelling or infiltration.** At first the follicular enlargement depends, it is true, upon abundant infiltration with plasma. If such a follicle be punctured, clear fluid escapes, and the follicle collapses. In the course of a few days, however, a marked increase in the number of lymph-cells themselves will have taken place. Now neither [illegible] from the follicles on puncture, nor do the follicles collapse. Upon section they present a whitish or yellowish aspect suggestive of the appearance of brain-tissue or boiled potatoes. Upon Peyer's patches the alterations described are often present only in places, so that some portions appear unchanged. Toward the ileocecal valve the follicular swelling increases, and occasionally the intestine in this situation is surrounded throughout its entire circumference with infiltrated follicular tissue. At times the morbid process extends to the solitary follicles of the mucous membrane of the large intestine. These are but rarely affected alone—so-called *colotyphoid.*

Microscopic examination of the diseased lymph-follicles discloses principally hyperplasia of the lymph-cells. Here and there large lymph-cells, with multiple nuclei—so-called typhoid cells—are found. The blood-vessels in the follicles appear dilated, in places filled abundantly with colorless blood-corpuscles, and their walls are in a state of vitreous swelling. Between the lymph-cells and in the lymph-vessels typhoid-bacilli occur, which generally lie close together in groups. Proliferative processes can be recognized also in the interstitial connective tissue. Further, the alterations described often extend beyond the actual area of a lymph-follicle to the adjacent intestinal wall.

In the course of the second week of the disease the stage of **exfoliation** develops. The superficial layers of the follicle undergo necrosis, partly in consequence of deficient blood-supply, and, in part, probably, also as an immediate result of the action of the typhoid toxins, and form eschars which are generally discolored yellow by fecal matter.

In the third week of the disease these eschars are thrown off, and beneath them the **typhoid ulcer** comes into view. In conformity with the distribution of the lymph-follicles, the long diameter of the resulting ulcers always corresponds with the long axis of the intestine. Often the floor of the ulcers is so clean that individual bands of the muscular layer of the intestine can be recognized without difficulty.

The fourth week of the disease includes the period of **cicatrization.** The cicatrix can be recognized for many years, occasionally throughout the whole of life, from the circumstance that, particularly in transmitted light, the intestinal wall appears translucent and attenuated in this situation, that the scar in the mucous membrane is frequently surrounded by dark-colored rings from the remains of previous hemorrhages, and that often glands are wanting in the cicatricial intestinal mucous membrane. Typhoid cicatrices do not give rise to cicatricial stenosis of the bowel.

The **mesenteric glands** undergo precisely the same alterations of at first catarrhal and subsequently hyperplastic swelling as the lymph-follicles of the intestinal mucous membrane. The lymphatic glands of the ileocecal chain, passing from the root of the mesentery toward the region of the ileocecal valve, are especially affected. At first the glandular tissue appears juicy, hyperemic, and grayish red in color, while subsequently it becomes anemic and medullary in appearance, and toward the end of the disease involution occurs, with fatty degeneration and absorption of the proliferated lymph-cells.

The *microscopic alterations in the mesenteric lymph-glands* agree with the corresponding changes in the lymph-follicles of the intestinal mucous membrane. In rare instances necrotic softening and rupture into the peritoneal cavity result, with consecutive perforative peritonitis.

The **spleen** is frequently enlarged to more than four times its normal size. The capsule of the spleen appears greatly distended, and upon section the organ exhibits a soft, almost diffluent tissue. In addition to multiplication of the splenic cells, cells are frequently found on microscopic examination containing several red blood-corpuscles or fragments of red corpuscles. Collections of typhoid-bacilli are encountered also in the spleen, and these may undergo multiplication even after death.

Occasionally the *peripheral lymphatic glands*, the *circumvallate papillæ of the tongue*, and the *tonsils* are the seat of swelling and medullary infiltration. The *bone-marrow* frequently appears hyperemic, and contains nucleated red blood-corpuscles in unusually large number.

The post-mortem alterations that are dependent upon the general infection are appreciable in the **dryness and the deep-red, ham-like color of the muscles of the trunk and the extremities.** Microscopic examination of the muscles discloses granular turbidity, fatty degeneration, and waxy degeneration. The last frequently is especially marked in the abdominal rectus muscles and the adductors of the thighs, and in these situations is appreciable as a yellowish salmon-like color. The **myocardium** is not rarely conspicuous on account of its flaccidity, and particularly its right half is frequently dilated. Microscopic examination discloses in the fibers of the heart-muscle alterations similar to those observed in the voluntary muscles. The liver-cells, the epithelial cells of the convoluted uriniferous tubules, the glandular cells of the stomach, the intestine, and the pancreas are the seat of cloudy swelling and fatty degeneration.

Treatment.—No *specific treatment for typhoid fever* has as yet been discovered. Blood-serum therapy has recently been attempted, but it has not yielded convincing practical results. Among the drugs recommended as specifics **calomel** (0.5—7½ grains—in a single dose) is deserving of the most confidence. If existing constipation is overcome by means of calomel at the beginning of the disease, the fever will occasionally be observed to subside speedily. [illegible] also, the subsequent course of the disease is notice- [illegible] Naturally, the results from the use of

In the majority of cases **expectant treatment** will suffice, and will include especially careful *nursing* of the patient. The *sick-room* should be as quiet as possible and free from bright illumination. In the summer it should be ventilated by opening the windows, while in the winter this should be done by opening the window in an adjoining room. It is advisable that access to the sick-room be gained through an anteroom, in order, so far as possible, to avoid draft. The *temperature of the sick-room* should be 20° C. (16° R.—68° F.). In the winter it is advisable to keep the air of the room moist by placing vessels containing water upon the stove or in the chimney. If circumstances permit, two *beds* should be available—one for the day and the other for the night. The head of the bed should be directed toward the window, in order that the patient be not annoyed and irritated by the sunlight. Care should be taken to keep the sheets smooth and free from folds, and to prevent the collection in bed of remains of food, especially dry bread-crumbs, as both of these are capable of exerting pressure upon the skin and may readily give rise to pressure-gangrene of the skin (bed-sores).

The patient should receive only *liquid nourishment*, particularly boiled milk, milk and coffee, milk and tea, and meat-broth with egg. Thirst may be relieved with pure spring-water, to which one-third of some mild red or white wine may be added. To prevent the accumulation of bacteria in the buccal cavity, and their entrance from here into the ducts of the salivary glands and inflammation of these glands, the *mouth* should be rinsed thrice daily with water or potassium chlorate (5.0 : 200). Between the hours of eight and ten and those of four and six the patient should be given a *warm bath* at a temperature of 35° C. (28° R.—95° F.), in which he should remain for from fifteen to thirty minutes. After the bath he is dried with slightly warmed towels, is supplied with a warm shirt, and is replaced in bed, which has been warmed with hot-water bottles. The bath refreshes the patient, cleanses the skin, and protects it from inflammation and gangrene.

Comatose patients should be proffered something to drink every half-hour. If the *lips* are excessively dry, they should be anointed morning and evening with oil of almonds in order to prevent the accumulation of sordes. In the case of comatose patients evacuation of the *bladder* at regular intervals should also be secured. If urine and feces are passed involuntarily, the patients should be kept dry and the skin be thoroughly washed with mercuric-chlorid solution (0.1 per cent.). If the skin becomes reddened at points exposed to pressure, and if gangrene as a result be threatened, the skin should be rubbed with dilute vinegar, alcohol, or lemon-juice, and the patient should be placed upon rubber cushions. It is advisable also to cover the skin smoothly with adhesive plaster, which can be shaped into the form of a Maltese cross by cuts in the four corners.

Drugs are not necessary in cases of typhoid fever pursuing a typical course. Should the patients, however, demand medicine, they may be given mineral acids, which relieve the sense of thirst and favor gastric digestion; as, for instance:

℞ Solution of phosphoric acid, 5.0 : 180 (75 grains : 5¾ fluidounces);
Sirup of raspberry, 20.0 (5 fluidrams).—M.
Dose: 15 c.c. (1 tablespoonful) every two hours.

After the patient has been free from fever for three days thin oatmeal-gruel may be tried, and after the lapse of a few days more scraped meat. Gradually the usual diet should be resumed. The patient may arise from bed after the fever has been absent for eight days, but this should at first be for only one or two hours, the period being gradually lengthened.

Symptomatic treatment should replace expectant treatment when individual symptoms acquire alarming intensity. It is my practice to make little use of antipyretics, and among these I prefer phenacetin (1.0—15 grains). I employ the remedies of this group only when the *temperature* persists above 41° C. (105.8° F.), or in the case of drunkards, the aged, and in the presence of disease of the heart and pregnancy when the temperature is above 39° C. (102.2° F.). In the presence of extensive and deeply seated *bed-sores* the patient should be kept continuously in a warm bath. If *intestinal hemorrhage* occur, an ice-bag should be applied over the ileocecal region, and solution of ferric chlorid be administered internally (10 drops every three hours in mucilage of salep). If symptoms of *peritonitis* or of *perforative peritonitis* appear, opium (0.02—⅓ grain—every two hours) should be administered; and if exhaustion be present, subcutaneous injections of camphorated oil should be made every two or three hours. In cases of perforative peritonitis operative treatment has also been successfully undertaken.

Great care should be devoted to the *prophylaxis of typhoid fever.* The urine and the stools should be disinfected with milk of lime (1 liter of calcined lime to 4 liters of water), the discharges being mixed with an equal amount of milk of lime and permitted to stand for two hours. The body-linen and the bed-linen of the patient should first be kept for twenty-four hours in a 5 per cent. solution of carbolic acid before being given to the laundress to be washed. The *clothing* should be disinfected by means of live steam.

Every community can protect itself most securely against typhoid fever by providing a perfect *water-supply,* if possible purified by means of sand-filtration and conveyed through water-tight pipes;

and, besides, by providing for the conveyance of *sewage* through water-tight conduits placed as remotely as possible from the water-pipes.

DYSENTERY.

Etiology.—Dysentery is principally an *endemic infectious disease of subtropical and tropical countries*, where it often prevails together with malaria. Foreigners, particularly explorers and colonists, who escape the one disease frequently fall victim to the other. In the temperate zone dysentery is one of the less common infectious diseases, which either occurs sporadically in strangers or occasionally acquires epidemic distribution through the latter. Epidemics of dysentery occur in temperate climates especially during the late summer and the autumn, and are not rarely favored by the ingestion of unripe vegetables and fruits; not that such indiscretions in diet alone excite dysentery, although they favor the activity of the germ of dysentery through mechanical and chemic irritation of the intestinal mucous membrane, especially that of the colon.

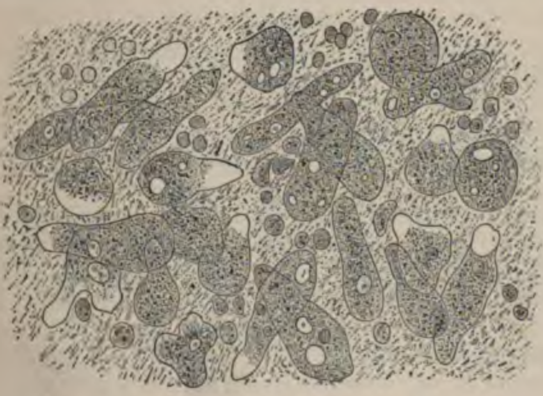


FIG. 70.—Amœbæ coli in intestinal mucus (after Lösch).

Little is known with regard to the *infective virus of dysentery*, and it would appear as if several pathogenic agents are capable of exciting the disease. In recent years *amebic dysentery* especially has attracted considerable attention, being present particularly in cases of tropical dysentery, Under these conditions the **Amœba coli** (Fig. 70) is found upon microscopic examination of the fresh stool, and it has been reported that severe enteritis can be excited by its introduction into the rectum of cats. Nevertheless, some clinicians still believe the ameba to be an accidental and insignificant feature of tropical dysentery. Nothing at all of a definite nature is as yet known with regard to *bacteria* as the cause of dysentery. It is certain, however, that the **infective virus is contained in the stools of dysenteric patients**, as the disease can be readily conveyed through carelessness or insufficiency in the disinfection of the stools, by means of bedpans, syringes, thermometers, or contaminated linen. The manner in which the infective material invades the human body is only a matter of hypothesis. Probably it gains entrance, as a rule, through the mouth or the nasopharynx, and is swallowed. Nothing definite is known with regard to conveyance through water.

Occasionally the germ of dysentery finds lodgment only in certain houses; thus, *house-epidemics* have been known to occur in barracks and in jails. In other instances an epidemic may extend throughout an entire community or throughout several neighboring communities. Dysentery has often prevailed widely in *times of war*, as the crowding in camps, deficient cleanliness, and indiscretions in diet must greatly favor the spread of the disease. Occasionally the disease has rapidly subsided with change in camp, and this has suggested the view that the virus of dysentery is contained in the soil.

Dysentery occurs equally in both *sexes* and at all *periods of life*. One attack of dysentery does not confer immunity from subsequent infection.

Anatomic Alterations.—Dysentery is attended with catarrhal, purulent, or necrotic **inflammation of the large intestine.** The inflammatory lesions are most marked in the rectum, and gradually subside toward the ileocecal valve. Only exceptionally do they extend to the mucous membrane of the small intestine.

In cases of *catarrhal dysentery* the mucous membrane of the large intestine is intensely reddened, and in many places more or less extensive extravasations of blood have taken place. The mucous membrane is greatly thickened and relaxed, and is covered with an abundant layer of mucus that is turbid in places. The solitary follicles of the mucous membrane are swollen and surrounded by a red garland of vessels. Destruction of solitary follicles also may take place, so that circular, crater-like, deep ulcers are encountered, often containing masses of mucus suggestive of the appearance of frog-spawn or boiled sago. Under such circumstances the designation *follicular dysentery* has been employed.

Purulent dysentery is attended with almost the same anatomic lesions, although they are often more pronounced. There is this difference, however, that the depositions upon the mucous membrane of the large intestine are distinctly of a purulent character.

Necrotic dysentery represents the highest grade of dysenteric inflammation of the bowel, and it results from catarrhal or purulent dysentery. The mucous membrane of the large intestine is, under such circumstances, converted into a grayish-green or greenish-black necrotic mass, which from its appearance has been designated moss-like, or, with reference to its distribution, geographic dysentery. The necrotic alterations are liable to be especially marked at the summit of the folds of the mucous membrane and along the bands of the colon. Also, the flexures of the colon (sigmoid, splenic, hepatic) generally exhibit especially marked necrosis, and this is attributed to the active mechanical irritation exerted by the intestinal contents. At the commencement of necrotic dysentery the necrotic areas upon the mucous membrane appear in the form of small spots and dots, and only subsequently do the diffuse lesions develop from the confluence of adjacent areas of necrosis. At the beginning the mucous membrane of the large intestine often presents an appearance as if it had been strewn with bran or ashes. If necrotic tissue is exfoliated, *dysenteric ulcers* result, often with deeply undermined margins. Occasionally adjacent dysenteric ulcers communicate with each other by means of submucous sinuses.

Microscopic examination of the intestinal mucous membrane discloses in cases of catarrhal and purulent dysentery marked dilatation and distention of the blood-vessels in the submucous and mucous coats, as well as marked accumulation of round cells in both layers of the mucous membrane. In the mucosa proper the round cells are often collected between the glandular tubes, in consequence of which the latter are often separated by considerable intervals. Often they are more or less completely destroyed. In cases of necrotic dysentery an exudate containing much fibrin, and therefore coagulable, has been poured out into the tissue of the mucous membrane, compressing the blood-vessels, and as a result interfering with the nutrition of the mucous membrane to such a marked degree that necrosis of the latter takes place.

The bodies of those dead of dysentery generally exhibit marked *emaciation*. The *spleen* often is unchanged.

Symptoms.—The *period of incubation of dysentery* is placed at from three to eight days. *Prodromes* are not rarely wanting, or consist in impairment of appetite, nausea, vomiting, or rumbling in the abdomen. Among the *objective symptoms of dysentery* the **evacuation of the bowels** and the **stools** are the most important. The patient has a desire to evacuate the bowels frequently, and this is not rarely so marked that the patient [illegible] to leave the bed-pan or the commode. This is designated [illegible] *tenesmus*. It is, therefore, not surprising that [illegible] discharge from the bowel may [illegible] The rectal tenesmus is often [illegible] **borborygmi**—and [illegible] will frequently be found that [illegible] tracted, and if a finger [illegible] tightly grasped by the [illegible] At the same time the [illegible] Likewise, almost every [illegible] contents reach the [illegible]

The *dysenteric stool* is thin, although at the beginning of the disease it is not rarely admixed with unusually hard fecal masses. In cases of catarrhal dysentery masses of mucus are encountered in the discharges, often spotted or streaked with blood. Shreds of mucous membrane also are encountered that resemble frog-spawn and boiled sago in appearance. This condition indicates the presence of follicular ulceration of the intestinal mucous membrane. Further, ingested starch-granules also may acquire a similar appearance, although these can be readily recognized from the circumstance that they assume a blue color on addition of tincture of iodin. The amount of fecal matter discharged during the day may reach 1000 c.c. Gradually the stools lose their fecal odor and acquire a stale odor suggestive of seminal fluid. In cases of purulent dysentery the stools contain pus. This often occurs in the form of fragments and shreds, which, on standing, are deposited at the bottom of the vessel and constitute the so-called *intestinal scrapings.* In cases of *necrotic dysentery* the stool contains much blood, and at times resembles rather a watery infusion of raw meat, at other times the rusty sputum of fibrinous pneumonia. Occasionally the stool acquires a blackish bloody appearance and an offensive odor. Under such circumstances the designation *putrid dysentery* has been employed. This generally terminates within a short time amid septic symptoms and rapid loss of strength. It is, therefore, known also as *septic* and *adynamic dysentery.* It is noteworthy that in all varieties of dysentery the stool contains a surprisingly large amount of albumin, so that it coagulates when heated. From this it may be comprehended that dysenteric patients rapidly become pallid and emaciated.

The **bodily temperature** remains unaltered or is but slightly elevated. The **pulse,** however, is generally increased in frequency. The **face** soon becomes sunken, and the **eyes** surrounded by gray shadows. The **tongue** is generally coated. Not rarely the **breath is fetid.** While the **appetite** is wanting, **thirst** is generally increased. Not rarely dysenteric patients are annoyed by **eructation, nausea,** and **vomiting.** The **abdomen** is frequently distended at first, but subsequently it becomes more and more retracted. The **left iliac fossa** is generally tender on pressure along the course of the sigmoid flexure, and on percussion it frequently yields a note of impaired resonance.

Dysentery may terminate in recovery within a few days or weeks, but in some cases the disease becomes *chronic.* It then occasionally persists for years, with frequent exacerbations and remissions. This condition is characterized anatomically by ulcerative changes in the intestinal mucous membrane—*chronic dysenteric ulcers*—which exhibit but little tendency to complete cicatrization. Amebic dysentery especially is characterized by this tendency to pursue a chronic course.

Among the *complications of dysentery* two groups can be distinguished. Those of the one depend immediately upon the intestinal alterations, and are rather *local in nature,* while those of the other are due to dissemination of the exciting agents of inflammation for considerable distances—so-called *dysenteric metastases.* When the inflammatory alterations have extended deeply into the intestinal mucous membrane, and have approached the serous layer, it may happen that this membrane likewise becomes inflamed, and in this way *circumscribed* or *diffuse peritonitis* will be developed. Occasionally intestinal perforation also occurs, and it may give rise to *sacculated* or *free perforative peritonitis.* These complications may, however, develop so insidiously as to escape detection. At times the cellular tissue surrounding the rectum undergoes inflammation—*periproctitis*—and suppuration may take place, with rupture of the pus either into the rectum or externally, or in both directions at the same time, in consequence of which an internal, an external, or a complete *rectal fistula* may develop.

Among the *metastatic complications of dysentery* two are worthy of special mention by reason of their frequency, namely, *abscess of the liver* and *polyarthritis.*

Abscess of the liver is not a rare complication, especially of tropical dysentery. Although it may begin while the dysentery is still present, it frequently becomes recognizable only after the dysenteric process has subsided, occasionally many months afterward, so that it then appears to represent rather a sequel of dysentery. Dysenteric amebæ have often been found in the pus from abscess of the liver, although some physicians maintain that the suppuration is due not to the amebæ, but to the pyogenic cocci associated with them.

At times *pulmonary abscess* develops in conjunction with abscess of the liver, and, under such circumstances, dysenteric amebæ have been found also in the sputum.

The *polyarthritis* often resembles acute articular rheumatism, and is attended with redness, swelling, and tenderness in various joints. At times suppuration takes place in some joints, with its sequels, viz., general septicemia or ankylosis.

Any viscus may become the seat of inflammatory alterations in the course of dysentery. *Neuritis* and *myelitis* may yet be mentioned, the former beginning, perhaps, in the immediate proximity of the rectum and extending to the spinal cord.

Of the *sequelæ of dysentery,* some have already been mentioned in the consideration of complications. Not rarely the *intestine remains sensitive* throughout life, with irregularity in the movement of the bowels. *Stenosis of the bowel* is a serious sequel, which may progress to *occlusion of the bowel* and terminate fatally amid symptoms of obstruction. Dysenteric cicatrices especially are noted for their great tendency to undergo contraction.

Diagnosis.—Dysentery is surrounded with no diagnostic difficulty in consequence of its peculiar symptomatology. Microscopic examination of the stools will decide if the case is one of amebic dysentery, although fresh stools must be utilized for this purpose, as the amebæ on standing lose their motility and are converted into circular bodies that are indistinguishable from round cells. In doubtful cases it is well to introduce a blunt glass tube into the rectum in order to obtain intestinal contents for microscopic examination. Rectal tenesmus and purulent or bloody stools occur also in association with **syphilis of the rectum, rectal polypi,** and **carcinoma of the rectum.** Examination of the rectum with the finger, the sound, or the speculum will generally afford information whether syphilitic stenosis of the rectum, a smooth and soft rectal polyp, or a hard, friable carcinoma of the rectum is present. Dysentery is distinguished from **hemorrhoids** by the fact that in the presence of the former dilatation of the hemorrhoidal veins is visible or palpable. Clinical and anatomic alterations in the intestine similar to those of dysentery occur as a result of arsenical or mercuric-chlorid poisoning. Here the previous history is decisive. In addition, necrosis and calcification of the epithelial cells of the convoluted uriniferous tubules are present in association with mercuric-chlorid poisoning.

Prognosis.—The prognosis of dysentery is grave, as, in the first place, the disease is of itself not free from danger, and, besides, complications not rarely occur by which life is greatly imperilled. Even after the lapse of years dangers occasionally set in if stenosis and occlusion of the bowel have developed.

Treatment.—An individual suffering from dysentery, even of the mildest variety, should be in **bed** and receive only **liquid nourishment,** especially mucilaginous decoctions, and old **red wine** for the relief of thirst. The Greek wines are especially to be recommended on account of the large amount of tannic acid they contain. Should exhaustion be present, mulled wine, consisting of a mixture of water and good red wine, cloves, and cinnamon, should be administered and be drunk as hot as possible. The abdomen should be covered with a **hot cataplasm.** So long as the stools contain solid masses **purgatives** should be administered; as, for instance:

℞ Castor-oil,	50.0 (1½ fluidounces).

Dose: One-half to be taken in black coffee.

Astringents—styptics—should be prescribed only after the stools have become completely liquid. Of all astringents, the powder of ipecac and opium is to be preferred, for ipecac-root has long been known also as dysentery-root:

℞ Powdered ipecac and opium, 0.3 (4½ grains);
Saccharin, 0.02 (⅓ grain).—M.
Make 10 such powders.
Dose: 1 powder every two hours.

Local treatment also has been attempted. Opium-suppositories are useful for the relief of troublesome rectal tenesmus:

℞ Powdered opium, 0.3 (4½ grains);
Cocoa-butter, sufficient to make 10 suppositories.
Dose: Introduce 1 suppository into the rectum daily.

Ice-water and solutions of tannic acid have been employed for intestinal infusion, and the results have been lauded. Infusions of quinin-solution (1 : 1000) have been recommended in the treatment of amebic dysentery. Intestinal infusions of a solution of carbolic acid or mercuric chlorid should be avoided, as intoxication may readily result. *Complications* and *sequelæ* should be treated in the customary manner.

The *prophylaxis of dysentery* is important. Individuals suffering from dysentery should be treated in isolated hospitals, or at least in isolated wards, and the *stools* should be disinfected with milk of lime. The bed-linen and the body-linen of the patient should first be placed for twenty-four hours in a 5 per cent. solution of carbolic acid before they are given to the laundress to be washed. When an epidemic of dysentery prevails the *diet* should be carefully regulated.

ASIATIC CHOLERA.

Etiology.—*India* is the home of Asiatic cholera, so that the disease has been designated also Indian cholera. It has frequently been conveyed to Europe through *human intercourse*, for the first time in 1830. Two routes are open for such transmission: either the *land-route—caravan-route*—over Southern Russia, or the *sea-route*, at the present day principally through the Suez Canal. From Europe Asiatic cholera has often been conveyed by ships to America, so that within a few years the disease became distributed over the entire earth as a *pandemic*. Since the year 1830 five such pandemic outbreaks have been observed. Further, even in India cholera does not prevail at all times with the same intensity; but also here it periodically attains the proportions of a severe epidemic.

The **exciting agent of cholera** is the cholera-bacillus, or the comma-bacillus, discovered by Robert Koch in 1884, and which can be constantly obtained from the intestinal discharges of cholera-patients (p. 410, Fig. 71).

The *comma-bacilli* of Koch are so named on account of their curved, comma-like shape. Nevertheless, not every curved bacillus is a comma-bacillus, for there are a large number of comma-bacilli, as, for instance, in deposits upon the teeth, in cheese, in water, etc., which exhibit the

closest resemblance to the comma-bacilli of Koch, so that great training and experience in bacteriologic methods are necessary in order to recognize cholera-bacilli with certainty. If they are examined in the living state in the hanging-drop, they will be found to exhibit active movement, which is effected by a terminal flagellum. The latter, however, will become visible only on the employment of special staining methods. The cholera-bacilli can be readily stained with aniline dyes, as, for instance, carbol-fuchsin. On treatment by Gram's method, however, they yield up the stain. There is, also, no difficulty in obtaining pure cultures of the cholera-bacilli. Inoculation-experiments upon animals were unsuccessful at first, because cholera-bacilli are exceedingly sensitive to the action of acids, and they were destroyed by the hydrochloric acid of the gastric juice. On the other hand, the experiments at once yielded positive results when the hydrochloric acid of the gastric juice was neutralized by a solution of sodium bicarbonate before the introduction of the cholera-bacilli, or when the bacilli were introduced directly into a loop of small intestine. Intentional infection through the swallowing of pure cultures of cholera-bacilli has also been successfully accomplished by a number of physicians in themselves, and unintentional infection likewise has taken place in a number of instances in laboratories in the manipulation of cholera-bacilli. From the biology of the cholera-bacilli it is important, for a comprehension of the clinical manifestations, that as yet no spores are known to exist, and that the bacilli offer but little resistance to drying. On moist surfaces, however, as, for instance, upon damp linen, in milk, on butter, cheese, and vegetables, they thrive quite well.

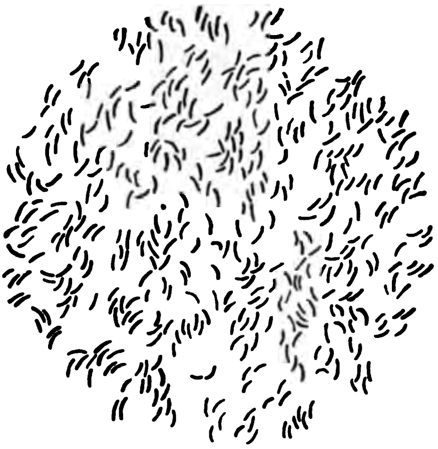


FIG. 71.—Cholera-bacilli: smear-preparation from a pure culture stained with carbol-fuchsin; oil-immersion; magnified 1000 times (from personal preparations).

The cholera-stool especially is capable of conveying infection from the cholera-patient, not rarely consisting of almost a pure culture of cholera-bacilli; and, in some instances, also, the *vomited matters*, which occasionally consist only of intestinal contents that have entered the stomach, and therefore also contain cholera-bacilli. Naturally, all articles are likewise infective that have been contaminated with the intestinal discharges or the vomited matters from cholera-patients, providing that the cholera-bacilli retain their vitality, or even undergo further development. Especial significance in this connection is to be attached to the dissemination of cholera through *water* contaminated by cholera-stools, and recently cholera-bacilli have been demonstrated in the water in several epidemics of cholera. Contamination of the water frequently results from throwing cholera-stools into streams whose water is used for domestic purposes; or from the infection of wells and water-conduits that are not water-tight by adjacent leaking cesspools, or from washing cholera-linen in public waters. In some instances infection takes place through *articles of food* that have been contaminated with cholera-stools and cholera-bacilli, as, for instance, through milk to which infected water has been added, through butter, cheese, or vegetables. Damp *linen* soiled by cholera-stools may also be the means of conveying the disease. Laundresses, therefore, are greatly exposed to the risk of cholera-infection. Recently attention has been more and more directed to the fact that *insects*, particularly flies, are capable of spreading the disease. It is well known that flies seek out malodorous materials, and therefore also cholera-stools. Should they then alight upon the lips of human beings, or upon articles of food, infection may readily take place. Cholera-bacilli have been demonstrated upon the legs and in the bodies of flies in cholera-rooms.

Inhalation of cholera-bacilli, with subsequent swallowing of the organisms, is possible, but probably occurs only exceptionally. In harbors and ports in which cholera has prevailed cholera-bacilli have been found in the sea-water, and it is, therefore, conceivable that water thrown into spray by waves may be inhaled by those residing on the banks of such streams. The inhalation of dry dust containing cholera-bacilli would scarcely be a source of danger, because the bacilli are readily destroyed by desiccation.

Inoculation through the rectum might occur if syringes or rectal tubes were employed in other cases without adequate disinfection after having been used upon cholera-patients; or if water-closets are used in which the seat or the paper has been soiled with cholera-stool.

That Asiatic cholera follows the lines of human intercourse, and is distributed thereby, is recognized, among other things, from the fact that in former years, before railroads and steamships were known, a much longer time was required for its spread to Europe than at present. A number of epidemics are also known that were spread by travellers from a cholera-place to remote cities, while all of the intervening places remained free from cholera. Thus, a few years ago, cholera was suddenly carried from Odessa to Altenburg. It is noteworthy that persons from places where cholera prevails may also convey the disease who are themselves free from cholera, for in such places cholera-bacilli have been found in the stools also of healthy persons. As may be understood, it is occasionally impossible, under the conditions last named, to discover the source of infection.

Certain conditions are calculated to favor the epidemic occurrence of cholera, and they have been designated also *contributory causes for the infection.* In the first place, experience teaches that cholera has first appeared with especial frequency in certain *commercial* and *maritime cities* of the European mainland, and this is probably dependent upon the active intercourse with foreign countries. The dirtier a place, the poorer its *water-supply* and *drainage,* the more favorable is the opportunity for the dissemination of cholera-bacilli and cholera. The *more low-lying portions of a city* are generally more severely attacked by cholera than the more highly situated, because the sewage flows from above downward. The *poorer classes of a community* are usually more seriously affected than the well-to-do, largely in consequence of a greater indifference to domestic order and cleanliness, and of a tendency to indulgence in inappropriate food. All *errors in diet* increase the danger of infection in an unmistakable manner. The number of cases is, therefore, often greater on Monday because of excesses in eating, and particularly in drinking, on Sunday. *Nervous disturbances* appear to be not without influence, and an unfavorable effect is, not without reason, attributed to the fear of cholera. As may be understood, all *aggregations of large numbers of human beings* favor the dissemination of cholera, and therefore annual fairs and festivals should be forbidden during the prevalence of an epidemic of cholera. The *season of the year* is of importance, as epidemics of cholera occur but seldom in the winter; most epidemics occur between the months of July and October. The *character of the soil* is probably of subordinate importance. Asiatic cholera may break out upon *ships,* in spite of earlier opinions to the contrary. The *weather* is probably without distinctive significance. The number of cases, it is true, has frequently been observed to diminish after persistent rain.

When an epidemic of cholera takes its origin from a single imported case, it has in a number of instances been observed that persons in the immediate vicinity (nurses, laundresses) were first attacked; then, in turn, the relatives of these, until gradually the circle of infection has extended. If an epidemic of cholera is dependent upon infection of the water-supply, it occasionally breaks out in an explosive manner throughout an entire large community. The *duration of the epidemic of cholera* generally extends from three to six months. As a rule, an epidemic progresses more rapidly than it subsides. *Secondary epidemics* also occur occasionally. Recovery from one attack of the disease does

not protect from subsequent attack. Sex and age have no influence upon the infection.

Symptoms.—The *period of incubation* of cholera may be as long as three days. Generally the disease sets in without *prodromes.* In accordance with the severity of the symptoms three grades of the disease are distinguished, and these are generally designated *cholera-diarrhea*, *cholerine*, and *asphyctic* or *algid cholera.*

Possibly still milder forms of cholera-infection occur, which are manifested only in cramps in the calves, borborygmi, and a sense of oppression.

Cholera-diarrhea frequently sets in during the early hours of the night. The patient, who had retired in perfect health, is awakened by griping and rumbling in the abdomen, with rectal tenesmus, and evacuates an unusually large amount of thin intestinal contents. Often he returns to bed after the first intestinal evacuation with a feeling of weakness; but in any event this does not fail to appear if, as is the rule, the stools are repeated five or six times before morning, and occasionally even oftener. The more frequently the bowels are moved the smaller is the amount of urine voided. The urine becomes dark, not rarely contains albumin and tube-casts, and it is particularly rich in indican. At the close of the act of micturition burning pain in the posterior portion of the urethra is not rarely appreciable. Cholera-diarrhea persists occasionally for but a single night, or it may continue for two or three days, seldom longer. Increased thirst then generally appears, the appetite is wanting, and the tongue becomes coated. The bodily temperature remains unchanged, but the pulse is increased in frequency. After recovery from cholera-diarrhea the patient often complains for days of great languor and exhaustion. Patients who pay no attention to the diarrhea are exposed to the danger that this may be converted into cholerine, or into asphyctic cholera. Cholera-diarrhea, as such, is scarcely attended with serious danger, except in reduced persons and in the aged.

Cholerine is characterized, in addition to the **diarrhea**, by a further symptom, namely, **vomiting.** The latter is attended with the expulsion at first of gastric contents, followed after a time by bilious, and finally by colorless matter. With proper care cholerine frequently terminates within a few days.

In cases of *asphyctic* or *algid cholera* **diarrhea** and **vomiting** persist as the principal symptoms, but, in addition, other disturbances make their appearance, which are dependent in part upon inspissation of the blood and enfeeblement of the circulation, and in part upon the profound intoxication of the body by cholera-toxins.

The *cholera-stool* generally soon acquires the characters of the feared *rice-water discharges.* In consequence of its frequency and [illegible] reaches 5 liters

[illegible] brownish and yel

[illegible] appearance. Th

[illegible] odor like that of

[illegible] large amount of

[illegible] and contains

[illegible] swollen

[illegible]

The **expired air** escaping through the nose and the mouth is conspicuous for its coldness. While the appetite is wholly wanting the patients are consumed by a burning and unquenchable thirst. The tongue and the mucous membrane of the mouth are dry and stick to each other. The **heart-sounds** are exceedingly faint, and particularly the first sound is frequently not audible at all. The **radial pulse** is often not palpable, whence the name asphyctic cholera; and if an artery be cut across only a few drops of blood often will escape. In the veins, also, only a small amount of thick blood is found. Occasionally **respiration** is deep, long drawn and labored, obviously in consequence of deficient pulmonary circulation. The voice is feeble, high, masked, and monotonous, and it is therefore designated also *choleraic voice.* Weakness of the muscles of the vocal bands is the cause of this condition.

The **abdomen** is, as a rule, slightly distended, and here and there loops of intestine can be recognized through the abdominal wall. On palpating the abdominal walls a sense of fluctuation is yielded from the presence of fluid in the intestine. Now and then rumbling in the abdomen can be appreciated. Spontaneous pain and pain on pressure are generally wanting. The **urine** is rapidly diminished in amount, and soon the secretion of urine is wholly suppressed. Such urine as is secreted contains indican in large amount, and frequently, also, albumin and tube-casts.

Although the skin feels cool, the **internal temperature of the body** is generally elevated, and the rectal temperature may be as high as 40° C. (104° F.) or higher. **Consciousness** is generally preserved until death occurs. In consequence of the rapidly developed physical debility the patients naturally enter into an apathetic state and become indifferent to their surroundings. Occasionally they are harassed by a sense of **fear, of oppression, and of palpitation of the heart.** Painful **muscular spasm**—choleraic cramp—constitutes an especially troublesome symptom, occurring periodically, most frequently in the calves, less commonly in the muscles of the arms, or even of the lower jaw. The patients groan and cry aloud, and clutch their muscles, which feel contracted with board-like hardness, and only after the lapse of several hours do the muscular contractions and the pain subside. Although formerly these manifestations were attributed to dryness of the muscles, toxic influences are at the present day held responsible for them. Similar muscular spasm is observed also in cases of diabetes, in which the assumption of autotoxic influences appears reasonable. Persons with asphyctic cholera frequently die within one or two days, although the disease may be protracted for a few days more.

Among the *anomalies of cholera, dry cholera* and *fulminating cholera* may be mentioned. Both varieties are characterized by their exceedingly rapid course, which may terminate within a few hours. Both represent the expression of an unusually profound intoxication with bacterial poisons (toxins). In cases of dry cholera, although frequently the patient may void no stool during life, on post-mortem examination the intestines will be found filled with fluid matter.

Cholera is rather free from *complications*. Occasionally *pericardial friction-murmurs* have been heard, resulting from unusual dryness of the layers of the pericardium. At times the stools become bloody and offensive in odor, and this is always a sign of unfavorable significance.

Should recovery from asphyctic cholera take place, this often occurs amid symptoms of the so-called *stage of reaction*. These are due in part to deficient renal activity and deficient elimination of urine, and in part to profound intoxication with bacterial poisons. The kidneys occasionally excrete small amounts of urine for days, and the fluid contains albumin, tube-casts, much indican, and frequently also a body capable of reducing an alkaline solution of copper, and which is generally held to be glucose. The longer the secretion of urine remains abnormal the less favorable is the outlook for recovery. The patient becomes comatose, with delirium, elevation of temperature, and dry tongue, and a roseolous exanthem frequently appears upon the skin, so that the entire clinical picture is suggestive of typhoid fever, whence the designation *cholera-typhoid* has also been employed. Occasionally the urea retained within the blood is eliminated with the sweat through the skin, and evaporation of the sweat may leave a whitish deposit of crystalline urea upon the skin—so-called *uridrosis*. Inflammatory processes in the various organs, venous and arterial thrombosis, and gangrene of the skin and the individual members also develop readily. Many patients who survive asphyctic cholera fall victims subsequently to the stage of reaction.

Diagnosis.—The recognition of cholera is possible with certainty only by *bacteriologic means*. In about half of the cases it is sufficient to obtain flocculi from the stools, spread them upon cover-slips, dry these by passing them through a flame, and stain them with carbol-fuchsin in order to find cholera-bacilli with certainty. In other instances, however, it is necessary to investigate a number of cultural and biologic peculiarities that Koch has described, and for the detection and recognition of which great skill and experience are required. It will, therefore, be well in all doubtful cases to submit such investigations to experienced hands, and to avail one's self of the services of a well-conducted bacteriologic institute. This is especially true of the first cases of cholera, as an incorrect diagnosis of cholera would cause great perturbation in a community, and failure to recognize a case of cholera promptly might result in incalculable consequences with regard to the dissemination of the disease, because the necessary measures of isolation and disinfection are neglected. The early and certain recognition of the first case of cholera especially is, therefore, of the greatest importance. The same clinical picture as Asiatic cholera gives rise to is encountered also in cases of **European cholera,** and in some instances of **poisoning,** especially with antimony and potassium tartrate, arsenic, mercuric chlorid, mussels, fungi, veratrum, colchicum. In the differential diagnosis the bacteriologic findings alone are decisive. Confusion is possible, further, with **incarceration of the bowel**; the various sites of hernia should, therefore, be carefully examined.

If the presence of Asiatic cholera is established, it can be readily determined from the symptoms whether cholera-diarrhea, cholerine, or asphyctic cholera exists.

Prognosis.—The prognosis of Asiatic cholera is in all cases grave, for there is no certainty that in spite of all precautions even mild attacks may not be converted into severe attacks. Naturally, the prognosis of cholera-diarrhea is more favorable than that of cholerine, and that of the latter better than that of asphyctic cholera. In general the mortality ranges between 50 and 60 per cent.

Anatomic Alterations.—The *choleraic facies* remains conspicuous even in the dead body. Often the **muscles of the extremities** are sharply outlined beneath the skin, and give the extremities unusual attitudes, which have been designated *fencing attitudes.* It is noteworthy that occasionally the muscles exhibit post-mortem contraction, so that even some of the fingers, and even entire extremities, may be engaged in movement as late as two and a half hours after death. Occasionally the body is said to have been found in an entirely different position than it had occupied, and this naturally has afforded some support for the tradition among the laity as to apparent death.

All of the internal organs exhibit pallor and dryness. The **serous membranes** (pleura, pericardium, peritoneum, meninges) feel as if smeared with soap. The **heart** contains only a small amount of inspissated blood. The pallid, bloodless **lungs** appear emphysematous. The **stomach,** but especially the **intestines,** contains an abundance of rice-water-like fluid. The epithelium of the intestinal mucous membrane is in many places detached, and floats in the intestinal contents. The mucous membrane appears hyperemic upon the summit of the folds and villi, especially in the ileum, but less so in the large intestine. Often the intestinal follicles are slightly enlarged. The intestinal serosa presents an actively injected appearance and a rosy discoloration. The **spleen** exhibits no peculiar alteration, nor does the *liver*, but the gall-bladder contains little bile of rather mucous consistency and grayish color. The **kidneys** are notable for their anemia and the flaccidity of their tissues.

On *microscopic examination of the viscera* the epithelial cells of the intestinal mucous membrane will be found in various stages of necrosis and degeneration. In the interstices of the intestinal glands, and between their epithelial cells and the membrana propria of the gland-tubules, the cholera-bacilli will be encountered. The latter are occasionally present, also, in other situations, as, for instance, in the biliary passages, the kidneys, the spleen, and the subarachnoid spaces of the brain. The blood-vessels of the intestinal mucous membrane and the serosa are dilated and greatly filled with blood. Between the glandular cells and in the submucosa abundant round-cell accumulation is evident. In the *kidneys* especially the often extensive epithelial necrosis in the convoluted uriniferous tubules will attract attention, resulting probably rather from the action of toxins than from circulatory disturbances.

Treatment.—Individuals with Asiatic cholera, even with the mildest form of the disease, should remain in **bed** until complete recovery has taken place, as otherwise a severe attack of cholera may readily develop. The **food** should be restricted to fluids, particularly tea, cooled coffee, and meat-soup with rice or sago. For the relief of thirst small bits of ice should be permitted to melt in the mouth, or small amounts of boiled, and subsequently cooled, water swallowed, and to which a few teaspoonfuls of cognac or half as much red wine is added. The ingestion of large quantities of fluid always increases the nausea and the vomiting. Should symptoms of collapse appear, mulled wine, eggnog, or champagne should be prescribed. The abdomen should be covered with a **hot cataplasm.** The treatment thus far outlined is applicable to all varieties of cholera. Among drugs, **preparations of opium** enjoy a not undeserved reputation in the treatment of *cholera-diarrhea* and *cholerine;* as, for instance:

℞ Tincture of opium,
Ethereal tincture of valerian, each, 5.0 (75 minims).—M.
Dose: From 10 to 20 drops every three hours.

In the treatment of *asphyctic cholera* the use of opium **has** rather been advised against in recent epidemics, and **calomel** recommended instead, for the purpose of rapidly expelling the cholera-bacilli from the intestine, and possibly also of destroying them. Nevertheless, I should suggest a combination of opium and calomel:

℞ Powder of ipecac and opium,	0.5 (7½ grains);
Mercurous chlorid,	0.1 (1½ ");
Sugar,	0.5 (7½ ").—M.

Make 10 such powders.
Dose: 1 powder every three hours.

To relieve the frequent vomiting, as well as the painful **muscular** spasms, **subcutaneous injection of morphin** should be employed:

℞ **Morphin hydrochlorate, 0.3 (4½ grains);**
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: From 4 to 8 minims subcutaneously.

Frictions of the skin with alcohol, solutions of mustard, camphor, or other spirituous solutions are less efficacious in the relief of the muscular spasm. If the body is cool, an effort should be made to render it warm by means of hot-water bottles, which may be placed in large number on either side of the body. Any bottle or jar may be used for this purpose, being filled with hot water and securely stoppered. Naturally, too great a degree of heat is to be avoided in order to prevent burning of the skin. Cholera-patients have also been placed for considerable lengths of time in **hot baths** at a temperature of 40° C. (104° F.), for the purpose of averting dangerous cooling of the body.

As an undeniably great danger to life resides in the loss of water from the body, efforts have been again made in recent epidemics of cholera to furnish the cholera-patient with considerable amounts of water through the skin, the blood-vessels, or the intestines. Thus, subcutaneous infusions of physiologic salt-solution (0.75 per cent.)—so-called **hypodermoclysis**—have been advised, the fluid being warmed and one or two liters (quarts) being permitted to flow beneath the skin upon the lateral aspect of the chest or the abdomen through a funnel and tube. Warmed physiologic solution of sodium chlorid has been employed also for **intravenous transfusion.** Although the patients improved temporarily, permanent results were generally wanting, and the mortality was not diminished. Solutions of tannic acid (tannic acid, from 3.0 to 10.0 : 2000; gum arabic, 30.0—1 ounce; tincture of opium, 20 drops, at a temperature of from 30° to 40° C.—86° to 104° F.) have been recommended for intestinal infusion—**enteroclysis.** V. Genersich permitted as much as 15 liters of solution of tannic acid (from 0.1 to 0.2 per cent.) at a temperature of 40° C. (104° F.) to flow into the bowel, so that the entire digestive tube was filled —a procedure that he has designated **diaclysmosis.**

No *specific remedy* for the treatment of cholera is known. Experiments with **blood-serum therapy** have been unattended with success. In the *stage of reaction* of cholera extensive use should be made of *hot baths.*

In the *prophylaxis* it is of great importance to recognize promptly the first case of cholera with certainty in order to institute the necessary isolation of the patient and disinfection of the stools, the vomited matters, and the linen. It is advisable to exercise sanitary supervision of travellers from places where cholera is prevalent, and to interdict the use of articles of food from such places. Complete quarantine of infected from healthy places would constitute a certain preventive measure, but it cannot thoroughly carried out without causing serious interference with travel and commerce. Supervision of commerce at the boundary is important. During the prevalence of cholera in India rigid sanitary scrutiny of vessels passing through the Suez Canal is necessary in order to prevent the introduction of the disease into Europe; but in Europe also, at times when cholera prevails, the movements of vessels upon the rivers should be placed under the supervision of the sanitary authorities, as cholera may be readily disseminated by this means. At times when cholera prevails all persons should avoid dietetic errors and excesses of every kind. Only boiled and subsequently cooled water should be drunk, and this may be made more palatable by the addition of lemon-juice.

Patients suffering from cholera can be best taken care of in special hospitals. Vomited matters and intestinal discharges should be disinfected with equal parts of milk of lime, and the linen should be immersed for twenty-four hours in a 5 per cent. solution of carbolic acid before being sent to the laundress to be washed.

The bodies of those dead of cholera should be wrapped in linen cloths saturated with a 5 per cent. solution of carbolic acid, and be placed in hermetically sealed coffins. The possessions of the patient and the sick-room should be disinfected in the customary manner.

Every community should endeavor to provide a perfect *water-supply* and *drainage*, for every deficiency in this connection may be followed by incalculable results at a time when cholera is prevalent.

EUROPEAN CHOLERA (CHOLERA MORBUS; CHOLERA NOSTRAS).

During the summer months attacks of a disease occasionally occur, at times in sporadic, at other times in epidemic, distribution, that resembles Asiatic cholera in all its manifestations from cholera-diarrhea to asphyctic cholera. One factor only, however, is never present in European or indigenous cholera, namely, the comma-bacillus of Koch. In all probability European cholera likewise is dependent upon bacteria, but these are as yet unknown. Death occurs but seldom, and as a rule only in reduced and aged persons. The *treatment* is the same as that for Asiatic cholera. Among the *causative factors* dietetic errors especially are to be mentioned, as, for instance, the ingestion of putrid water, spoiled meat, and unripe fruit and vegetables. In the *differential diagnosis* the forms of poisoning mentioned on p. 417 must be taken into consideration, in addition to Asiatic cholera.

The designation *Weil's disease* has been applied to a peculiar form of *infectious jaundice*, which generally occurs in the summer months. It sets in with chilliness or a chill, which is followed by elevation of temperature to 40° C. (104° F.) and above. The patient complains of *pains in the muscles*, particularly in the calves, becomes comatose and delirious, and presents *jaundice* and *enlargement of the liver and the spleen*. The *urine* contains biliary coloring-matter, generally albumin, not rarely, also, blood and tube-casts. The *stools* are not rarely free from biliary coloring-matter. In the course of one or two weeks the temperature gradually declines, but relapses frequently occur. The disease has often been observed in butchers, although it has occurred also after the swallowing of sewage and after bathing in the Danube near Ulm. The *causative agent* of the disease is unknown. On *post-mortem examination* granular clouding and fatty degeneration have been found in various organs. Recovery occurs as a rule. At the beginning of the disease a large dose of *calomel* (0.5—7½ grains) has been recommended; but subsequently the treatment should be confined to regulation of the diet.

VI. INFECTIOUS DISEASES ATTENDED WITH LOCAL LESIONS OF THE SEXUAL ORGANS.

GONORRHEA.

Etiology.—The designation gonorrhea is applied to every variety of inflammation that is excited by definite micro-organisms, namely, the **gonococci** discovered by Neisser in 1879. The condition most frequently encountered is gonorrhea of the urethral mucous membrane, so that it has become customary to imply gonorrhea of the urethral mucous membrane when the designation gonorrhea is employed. Gonorrhea of the rectal mucous membrane, of the cervix uteri, of the conjunctiva, and of the buccal and nasal mucous membrane also occurs. In the following description reference will be made exclusively to gonorrhea of the urethral mucous membrane.

Gonococci can be readily isolated from the usually purulent secretion from the mucous membranes. A drop of pus, for instance, from the infected urethra is placed upon a cover-glass, upon which a second cover-glass is gently permitted to fall, so that the pus shall be distributed in a thin layer between the two glasses. Then the cover-slips are drawn apart and exposed to the air with the surface of pus upward until dry. Now each slip is held between the thumb and the index-finger and is drawn with moderate celerity through a gas-flame or a spirit-flame for from six to ten times. A few drops of carbol-fuchsin, or of solution of gentian-violet or of methylene-blue, are poured upon the surface of the pus, and the cover-slip is rinsed after five minutes in water, is again dried by being passed several times through a flame, and is then embedded in a drop of xylol-Canada balsam that has been placed upon a slide. The preparation is now ready for microscopic examination.

Gonococci are generally collected together in *groups*, and are in part free and in part contained *within cells* (p. 422, Fig. 72). It is especially this enclosure within cells, whose nuclei appear eroded, that is characteristic of gonococci. On more careful inspection it will be found that the gonococci are *hemispherical* or *biscuit-shaped in form*, and generally are arranged in pairs as *diplococci*, being separated from each other by only a delicate interval. Tinctorially they are distinguished by the circumstance that they are decolorized by Gram's method. *Pure cultures* cannot be readily obtained. The organisms have been cultivated upon solidified human blood-serum and upon agar to which ascites-fluid or human urine has been added. Also, the white of plovers' eggs has been recommended as a culture-medium for gonococci. *Inoculation-experiments with pure cultures of gonococci* have been repeatedly performed successfully upon human beings.

Gonorrheal urethritis is generally a result of *sexual intercourse* with an individual suffering from gonorrhea. Infection is acquired with especial frequency in public houses of prostitution in which the women inmates are not examined at all, or with but insufficient care and frequency, for the presence of gonorrhea, and in case of disease are not at once placed out of service. A single woman in a house of prostitution often cohabits with more than ten men in the course of a night, all of whom may acquire gonorrhea. Intentional prolongation of the sexual act increases the risk of infection, because under such circumstances the infective secretion may penetrate the more readily and the more deeply into the urethra. The mode in which infection is acquired explains why the victims are most commonly unmarried and young men. Gonorrhea occurs in children, almost exclusively in girls, who have been raped, or if children occupy the *same bed* as an adult suffering from gonorrhea, so that transfer of the discharge may readily take place.

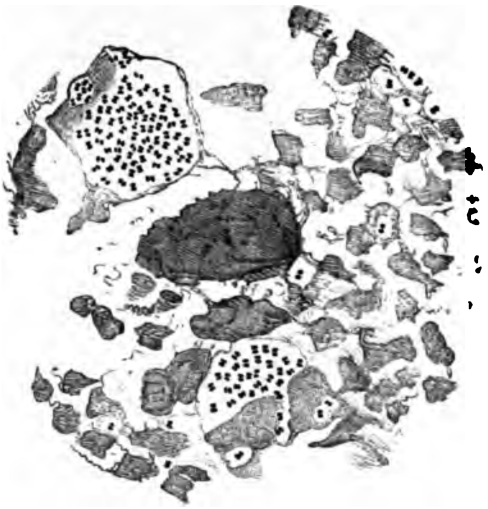


FIG. 72.—Gonorrheal pus containing gonococci; dry preparation stained with carbol-fuchsin; oil-immersion; magnified 1000 times (personal observation).

Inoculation may be effected by means of *dirty hands and instruments* to which gonococci are adherent. In women gonorrhea of the rectal mucous membrane occurs occasionally in consequence of overflow of gonorrheal discharge from the vagina upon the perineum. In some cases, it is true, gonorrhea of the rectum is a result of unnatural cohabitation. The same statement is applicable to gonorrhea of the buccal mucous membrane. One who has had an attack of gonorrhea is readily attacked again on exposure to a source of infection. Some individuals exhibit a greater predisposition to infection than others, which is illustrated, among other things, by the fact that if several men cohabit with the same woman successively, only a few will be attacked with gonorrhea. Occasionally *other venereal disease* may be present in the same person in association with gonorrhea (soft chancre and syphilis). Infection may take place either through a single cohabitation or after successive cohabitation with several persons.

Symptoms, Prognosis, and Diagnosis.—The *period of incubation of gonorrhea* is often from twelve to twenty-four hours. All statements referring to the lapse of weeks are worthy of little credence and usually originate from the circumstance that the patient endeavors to suppress his sense of shame in the presence of the physician by referring his folly as far back as possible. Gonorrhea probably always begins as *acute gonorrhea*, which, however, frequently passes into *chronic gonorrhea*. Gonorrhea in the male and in the female will be considered separately in the following description.

Acute Gonorrheal Urethritis in the Male.—In most patients attention is attracted to their disease by the gluing together of the *lips of the urethra* by secretion, so that the stream of urine in the act of micturition must first overcome an obstruction before it can be expelled. Soon tickling, but in a little while painful, sensations become appreciable after the act of micturition, obviously in consequence of irritation of the inflamed urethra by the urine. In addition there is increased vesical tenesmus. Gradually a progressively increasing discharge takes place from the urethra, at first mucopurulent, but soon becoming wholly purulent and containing gonococci, together with generally polynuclear round cells and pavement epithelial cells from the urethral mucous membrane. The discharge often soils the linen, giving rise to greenish-yellow stiff stains, and it is highly infectious, so that it may be conveyed by means of the fingers to the conjunctiva and give rise to the gravest forms of ocular inflammation (blennorrhea, diphtheria) and culminate in blindness. Left to itself, the urethral discharge diminishes in the course of from four to six weeks, becomes more mucoid, and eventually ceases spontaneously.

Unfortunately, urethral gonorrhea is frequently attended with *complications*, which at times result from an improper mode of life, at other times from inappropriate treatment, and occasionally also are dependent upon the posture assumed by the patient. They are in part the result of reflex irritation, in part of inflammatory origin. In the latter event the inflammatory process may in turn have extended directly to adjacent structures or have developed in remotely situated organs—so-called *gonorrheal metastases*—and be due to the activity of gonococci, streptococci, or other phlogogenic bacteria. Among the complications of reflex origin are *increased sexual desire* and *painful erections*. The latter occur especially when the patient lies upon his back, and cause disturbance of sleep. Occasionally erections of the penis take place only in its posterior portion, while the anterior portion remains flaccid and hangs downward like a flail from its handle. This condition, which is a source of great anxiety to the patient, is attributed to the formation of thrombi in the blood-spaces of the cavernous body, preventing distention of the most anterior alveolar spaces with blood in the process of erection. Often this incomplete erection of the penis is attended with intense pain, as if a strand of catgut were being drawn through the organ, whence the designation *chorda penis*.

Among the minor complications are increased vesical tenesmus and *increase in the amount of urine.*

Among the *inflammatory complications in adjacent organs* are inflammation of the glans or the prepuce, or both together—*balanitis, posthitis, balanoposthitis.* This occurs with especial frequency in men possessing a contracted prepuce, so that gonorrheal pus may become readily imprisoned in the preputial sac and give rise to inflammation of the glans and the prepuce. The wearing of tight trousers and too long-continued and active physical exertion favor the development of these complications. Posthitis is often the starting-point for an inflammatory *phimosis*, the prepuce becoming so greatly swollen that it can be retracted over the glans only with a certain degree of violence, if at all. When the retraction has been effected the prepuce not rarely constricts the base of the glans tightly and it cannot be brought forward over the glans again. In this way the phimosis is converted into a *paraphimosis*—*Spanish collar*—and this is attended with the danger that in consequence of tight constriction of the glans the supply of blood becomes so deficient that gangrene of the glans and also of the constricting ring of prepuce may result.

In rare instances the inflammation of the mucous membrane of the urethra extends to the connective tissue surrounding the urethra, with the development of *periurethritis*. Painful thickening of the urethra is the principal manifestation. Should suppuration occur, the pus may burrow its way either into the urethra [illegible] in both directions simultaneously, and there will result, accordingly, an incomplete internal or external or a complete *urethral fistula*. On the whole, it is rare for one of the two lymphatic vessels in the median line on the dorsum of the penis to become inflamed—*lymphangitis*—with the formation of a hard painful cord. Here and there nodular swellings may appear—*bubonuli*—which at times undergo suppuration and rupture externally. Even the *inguinal lymphatic glands* occasionally are affected in association with the urethritis, becoming swollen and painful—*acute inguinal bubo*—although they rarely undergo suppuration.

One of the most frequent inflammatory complications of gonorrhea is *inflammation of the epididymis*—*gonorrheal epididymitis*. As acute gonorrhea commences in the anterior portion of the urethra and only gradually extends to the posterior portion (membranous and prostatic portions), it can be understood that epididymitis generally does not develop before the third or the fourth week of the disease, occasionally even later. The development of epididymitis is favored by too irritating urethral injections and by traumatism, including the compression exerted by tight trousers, standing or walking for too long a time, failure to use a suspensory bandage, and permitting the testicle to hang dependent in going about. Generally but one testicle is involved in the inflammatory process.

The disease is characterized by pain, which is generally so unbearable that the patient is unable to go about. The involved half of the scrotum is not rarely enlarged to more than the size of a fist, presents a reddened, smooth, and edematous appearance, feels hot, and has become adherent to the vaginal tunic of the affected testicle. Palpation of the diseased testicle and epididymis is extremely painful. The epididymis often cannot be felt at all at first, because the cavity of the vaginal tunic proper is filled with a large amount of serous fluid. Fever is not rarely present. Occasionally vomiting and obstinate constipation occur, so that the clinical picture is suggestive of strangulated hernia. The swelling, together with the remaining inflammatory manifestations, subsides but slowly. Indurations in the epididymis persist throughout life with marked frequency. After bilateral disease *sterility in the male* not rarely results, as the vas deferens on each side may be occluded. Occasionally also infection with tubercle-bacilli may take place subsequently, and to the *tuberculosis of the epididymis* urogenital tuberculosis may be superadded. The vas deferens is not rarely involved in the inflammation of the epididymis—*gonorrheal deferentitis*. Under such circumstances the vas deferens will be found to be thickened, often presenting nodular enlargements, hard and exceedingly tender on pressure.

Occasionally inflammation of the *prostate gland*, the *seminal vesicles*, or of *Cowper's glands* occurs. All of these inflammatory processes give rise to pain in the perineum, and are not rarely attended with high fever and chills. In the presence of *gonorrheal prostatitis* the finger introduced into the rectum will feel the enlarged and exquisitely sensitive prostate in front of the wall of the rectum. Should suppuration take place, the pus may rupture into the rectum or the bladder, after which natural recovery is possible. In some cases, however, general septicemia develops, and not rarely terminates fatally. In the presence of *inflammation of the seminal vesicles—gonorrheal spermatocystitis*—examination with the finger in the rectum discloses an indurated and painful swelling to the side of the prostate gland. The modes of termination are the same as those for prostatitis. *Inflammation of Cowper's glands* is attended with painful induration just behind the scrotum to one side of the median line of the perineum. Should suppuration occur the pus may readily rupture externally.

Gonorrheal urocystitis develops frequently, and often, like epididymitis, results from unduly irritating injections or mechanical treatment—catheterization. Vesical tenesmus, pain in micturition, highly mucous, frequently purulent and bloody urine are its most conspicuous manifestations. Occasionally the inflammatory process extends along the ureters to the pelvis of the kidneys and *gonorrheal pyelitis* may develop. Even the kidney itself is occasionally involved in the inflammatory process, so that the symptoms of *acute hemorrhagic nephritis,* occasionally also those of *abscess of the kidney,* may be encountered. The latter are of serious import and generally terminate fatally from general septicemia.

Among the so-called *gonorrheal metastases,* namely, inflammatory processes in remote organs, *gonorrheal arthritis* should be mentioned first. This occurs most frequently in the form of polyarthritis, less commonly in that of monarthritis. The large joints of the extremities, especially the knee and the ankle, are involved with especial frequency. The clinical picture often resembles that of acute articular rheumatism, and, together generally with slight febrile movement, is attended with swelling, redness, and pain in the affected joints. It may also happen that the pain in the different joints varies and flies from one to another. Gonococci have in a number of instances been found in the articular exudate. The disease exhibits a marked tendency to pursue a protracted course, offers great resistance to salicylic acid and allied drugs (so that to a certain degree the gonorrheal origin of an arthritis may be suspected from failure of the drugs named), remains confined for a particularly long time to a single joint, and is frequently followed by stiffness of the joint and wasting of the adjacent muscles, particularly the extensors. Occasionally symptoms of verrucose or ulcerative endocarditis are superadded. It may also happen that suppuration takes place in some joints, and that septicemia or permanent ankylosis occurs as a complication. At times the *arthritis is purulent* from the outset. Gonorrheal arthritis is sometimes manifested also by the development of *hydrarthrosis*, often with exceedingly slight pain. Further, the joints may be extremely painful on active and passive movement, without presenting any other alteration—*dry gonorrheal arthritis.* As a result, the joints at times become deformed and movable with difficulty if at all—*deforming gonorrheal arthritis.*

As compared with gonorrheal arthritis, other metastases of gonorrhea occur much less commonly. Among them, *myelitis* and *abscess of the spinal cord, neuritis, neuralgia*, especially *gonorrheal sciatica, verrucose* and *ulcerative endocarditis, pleuritis, myalgia, iritis*, and *iridocyclitis* may be mentioned. Among the *sequelæ* of acute gonorrheal urethritis the formation of *acuminated condylomata* may yet be mentioned, developing sometimes at the urethral orifice, at other times on the frenulum or in the coronary sulcus, and occasionally forming immense growths resembling a cock's comb or cauliflower. They are due to the irritation of the skin by the gonorrheal pus, causing abnormal hyperplasia of the papillary bodies and the epithelial cells.

The *diagnosis* of acute urethral gonorrhea in the male is easy, as almost every purulent discharge from the urethra, particularly in a young man, is of gonorrheal origin. In addition, the presence of *gonococci* can easily be demonstrated in the pus.

In recent cases of acute urethral gonorrhea the pus generally flows spontaneously from the urethra, providing that the patient has not a short time previously passed urine, and thereby washed the pus out of the urethra. Otherwise continued pressure should be made with the finger upon the urethra from the most posterior portion of the urethra forward. In some individuals active secretion takes place in the anterior portion of the urethra on sexual excitement, causing the lips of the urethra to stick together and possibly leading an inexperienced observer to believe that gonorrhea is present. The secretion is derived from the glands of Littre in the urethral mucous membrane. The differential diagnosis is easy, as the secretion takes place only on sexual excitement, and is but transient. It is not purulent, and it contains no gonococci. In some men a discharge from the urethra occurs after sexual intercourse with women suffering from irritating discharges, as, for instance, too soon after menstruation or in the presence of carcinoma of the uterus. A transient discharge from the urethra also takes place at times after *catheterization* or when a *stone is impacted* in the urethra, but this secretion likewise is free from gonococci, in addition to the fact that the history will point to the antecedent etiologic factors. Occasionally a discharge from the urethra takes place in the course of *gout*—so-called gouty gonorrhea; gonococci are wanting also under such conditions. Purulent discharges from the urethra may occur in individuals with *sarcoma* of the penis, although neoplastic nodules may then be palpable. It should not be forgotten that in the presence of hard and soft chancres in the urethra—*larval chancre*—a purulent discharge may take place from the urethra that may readily give rise to confusion with gonorrhea. Gonorrhea is distinguishable from *spermatorrhea* and *prostatorrhea* by the presence in the former of spermatozoa and in the latter of spermin-crystals, but never of gonococci.

The *prognosis* of acute urethral gonorrhea in the male is good, providing that the patient carefully adheres to the necessary methods of procedure. Unfortunately, only a few patients are willing to submit to this, but continue in the pursuit of their work in the usual manner in order not to excite the attention of their friends. Evil consequences from such an injudicious course are often not wanting. Complications readily occur, and, above all, acute urethral gonorrhea becomes transformed into the chronic variety, which, unfortunately, is frequently not amenable to treatment and may prove a source of unhappiness not alone to the patient, but often also to his wife and consequently to his entire family. That urethral gonorrhea may under such circumstances become a serious disease many patients learn to their sorrow only too late. It is clearly the duty of the physician to undeceive his patient in the belief that acute urethral gonorrhea is a harmless disorder that under all circumstances can be readily cured within a short time.

Chronic gonorrheal urethritis in the male results from acute gonorrhea of the urethra. It develops especially if an acute gonorrhea is not treated carefully enough, and for a sufficiently long time, or if renewed infection is acquired before the preceding one has been completely cured. The constitution of the patient likewise appears to be not without influence. In the presence of chronic gonorrheal urethritis there is not a constant discharge from the urethra; the urine generally contains the products of inflammation only during micturition, and these appear in the form of yellowish, more or less resistant shreds or rather mucopurulent filaments—so-called *gonorrheal threads*. On microscopic examination these filaments will be found to consist principally of polynuclear pus-corpuscles, among which epithelial cells from the urethral mucous membrane are here and there distributed. The latter are frequently in a state of swelling and necrosis. Gonococci may be wanting in the filaments either at times only or constantly. Cases of the latter kind, in which the diagnosis can be made with certainty only after observation continued for a considerable length of time, are not a source of danger with regard to the spread of the disease. Gonorrheal threads are generally present in especially large number in the morning-urine, because there has been opportunity for them to collect in the urethra in considerable number during the night. Frequently the lips of the urethra become glued together during the night, so that in the act of micturition the stream of urine first encounters an obstruction that generally is easily overcome. Also, a drop of mucopurulent secretion can at times be expelled on pressure—the so-called *morning-drop*. Occasionally some gonorrheal filaments exhibit blood-streaks, indicating the presence of *ulcers in the urethra*. Symptoms indicative of stricture of the urethra (feeble stream, with a slight curve, incontinence of urine, great straining of the abdominal muscles in the act of micturition) are also present at times. Naturally, examination of the urethra with a bougie or a silver catheter will not be neglected under such circumstances, as the cure of chronic gonorrhea cannot be expected until the stricture of the urethra has been relieved.

Chronic gonorrhea is always a most obstinate disorder, and many patients are never entirely freed from it. The disease not rarely exerts an unfavorable influence upon the *mental state*. The patient provides himself with special glasses for the reception of the urine, and he carefully counts and records the number of gonorrheal filaments present, practises expression of the penis every morning in order to obtain a larger number of threads from the urethra, changes from one physician to another, and so on.

Chronic gonorrhea is occasionally attended with the same *complications* as acute gonorrhea, although, on the whole, they occur less commonly. Urocystitis, even nephritis, originating without apparent cause, monarthritis and polyarthritis of chronic and obstinate course, etc., are frequently dependent upon chronic gonorrhea. Not rarely chronic gonorrhea is converted again into acute gonorrhea. Such an event may occur as a result of renewed infection, after the introduction of sounds and catheters into the urethra, and occasionally after seminal emissions and non-infectious sexual intercourse.

The *diagnosis of chronic gonorrhea* is easy, as gonorrheal threads can be detected without difficulty at the bottom of a glass filled with urine or on agitation. If the threads are streaked with blood, this will be indicative of the presence of ulcers in the posterior portion of the urethra, while obstruction to the discharge of urine points to stricture of the urethra, and this can be definitely confirmed without difficulty with the aid of a bougie or a catheter. *Endoscopic examination* of the urethra will yield the most reliable information with regard to the anatomic alterations in this canal.

In the presence of acute as well as of chronic gonorrheal urethritis a distinction must be made between *gonorrhea of the anterior* and *that of the posterior portion of the urethra*, accordingly as the bulbous portion or the prostatic and membranous portions of the urethra are involved in the inflammatory process. In the differential diagnosis the *two-glass test* has been recommended. In the act of micturition one-half of the urine is received into one and the other half into another vessel. In the presence of inflammation in the anterior portion of the urethra only the urine contained in the first glass will be rendered turbid by gonorrheal filaments, while that contained in the second remains clear, because the urethra has been washed clean by the urine first voided. Should, however, the posterior portion of the urethra be the seat of the inflammatory process, the urine passed into the second glass also will be rendered turbid from the presence of gonorrheal filaments, because these have found their way backward into the bladder and render the urine in this viscus uniformly turbid. The observation is, however, by no means certainly conclusive; especially in the presence of cystitis both specimens of urine will be turbid.

The *prognosis of chronic gonorrhea* is serious from the fact that the disease proves incurable with exceeding frequency. It is the cause of disaster in many families, as men may infect their wives, and frequently render them permanent invalids.

Acute gonorrhea in the female involves especially the urethra and the mucous membrane of the neck of the uterus. Gonorrhea of the vaginal mucous membrane appears to occur only in young women. In the same way as in men a purulent discharge containing gonococci takes place from the urethra either spontaneously or on pressure, and drops of pus can also be seen escaping from the external orifice of the uterus. The mucous membrane of the vaginal portion of the uterus often is deep red, almost bluish red in appearance, and here and there superficial loss of tissue has taken place—erosions—with especial constancy at the external orifice of the uterus. The mucous membrane of the vagina also is frequently the seat of swelling and marked redness. Purulent secretion containing gonococci is generally derived from the cervical canal of the uterus. The purulent discharge often escapes from the vaginal orifice and moistens the labia, which are glued together by inspissated pus. Frequently they also become inflamed and increased in size, and they acquire a markedly reddened appearance. They are not rarely the seat of erosions usually of a superficial character.

Women with acute gonorrhea complain principally of burning and pain in the act of micturition; frequently there is increased vesical tenesmus. The patients are conscious of a disagreeable sense of increased heat in the genitalia, of tickling that is readily transformed into pain, and not rarely also of increased sexual desire. The symptoms are aggravated by standing and walking. At the same time the inner surfaces of the thighs are easily moistened with the gonorrheal discharge, and there result here inflammatory lesions and epithelial excoriations.

Such *complications* as may arise are about the same as those that attend acute gonorrhea in the male. Instead of epididymitis *gonorrheal Bartholinitis* occurs, which is generally unilateral. The patients under such circumstances complain at first of severe pain in one labium, which becomes greatly swollen and presents a reddened appearance. Fluctuation develops, and eventually the pus ruptures internally or externally. Occasionally the suppuration persists for some time longer. At times gonorrhea extends to the mucous membrane of the body of the uterus and the oviduct and to the peritoneum, with the development of circumscribed or diffuse *peritonitis*.

Acute gonorrhea in the female also has a marked tendency to be converted into *chronic gonorrhea*. Should pregnancy occur, the newborn child may readily acquire a *conjunctival blennorrhea* in the act of parturition, and this may seriously threaten the integrity of the eye, or *puerperal septicemia* not rarely develops in the course of the puerperium.

The *diagnosis* of acute gonorrhea in the female is not attended with any considerable difficulty. In the police-inspection of prostitutes it should be borne in mind that the women are in the habit of passing urine shortly in advance of the medical examination in order that the urethra may appear free from pus, and that they are able with great skill to wipe away and remove the pus with their shirt. The presence of gonococci in the purulent discharge from the urethra can be readily detected; on the other hand, this is far more difficult with regard to the discharge from the neck of the uterus and the vagina, as this of and in itself contains cocci and bacilli in large number. In the differential diagnosis by bacterioscopic means it should be noted whether diplococci are contained within round cells, and whether they can be decolorized by the method of Gram.

The *prognosis* of acute gonorrhea in the female is favorable in so far as there is no immediate danger to life, but it is rendered more serious from the fact that occasionally, particularly when there is marked involvement of the neck of the uterus, complete recovery cannot be effected, and that with the occurrence of pregnancy and parturition disagreeable and dangerous complications may arise.

Chronic gonorrhea in the female is attended with a constant discharge from the genitalia, particularly from the neck of the uterus. Women who were healthy at the time of marriage may be infected by their husbands suffering from gonorrhea at this time, and discharges occur that had never before been present. Often symptoms of acute gonorrhea have preceded, but not rarely these are so slight that the patients, generally wholly unfamiliar with the subject of venereal diseases, believe themselves to have presented the symptoms of chronic gonorrhea from the outset. Then discomfort and pain in the lower portion of the abdomen gradually develop, miscarriages occur, endometritis and peritonitis readily appear as complications of labor; other women are attacked with chronic endometritis of apparently spontaneous origin, with purulent salpingitis, perimetritis or parametritis, or with diffuse peritonitis. Suppuration in the ovaries also occurs occasionally. It is remarkable how common chronic gonorrhea is in married women, and to how many marriage brings loss of health.

Naturally, chronic gonorrhea is especially common in prostitutes, who, in turn, constitute a source for the infection of large numbers of men with acute gonorrhea, for the discharge of chronic gonorrhea in women gives rise in turn to the symptoms of acute gonorrhea in healthy men. Among the sequelæ, especially of chronic gonorrhea, *acuminated condylomata* may be mentioned which occasionally present on the labia the appearance of cauliflower-like masses as large as a fist.

While the *diagnosis* of acute gonorrhea is more difficult in the female than in the male, the difficulty is even far greater with regard to chronic gonorrhea. The mucous membrane of the neck of the uterus and the vagina frequently exhibits but slight alterations. Erosions or ulcers are often found at the external orifice of the uterus, from which there flows a mucous, generally a mucopurulent, discharge, whose gonorrheal nature will be established by the demonstration of gonococci in it. This demonstration is, however, often exceedingly difficult, because all of the discharges from the female genitalia contain innumerable bacteria in the form of cocci and bacilli, and among which the differentiation of gonococci is by no means easy.

The *prognosis of chronic gonorrhea in the female* is not favorable. Complete recovery is not rarely attended with almost insurmountable difficulty, particularly if the lining membrane of the cavity of the uterus is also involved in the gonorrheal inflammatory process. Many women fail gradually, disappointed in the anticipated happiness of marriage, while others fall victim to grave complications if their hopes of maternity are realized.

Anatomic Alterations.—The anatomic alterations to which gonococci give rise in the urethral mucous membrane and the conjunctiva have in part been studied by experimental methods. It has under such circumstances been observed that the gonococci first undergo multiplication upon the surface of the mucous membrane, then penetrate deeply between the cement-substance of the epithelial cells, especially become more numerous beneath the epithelial layer, and find their way into the lymph-spaces of the submucosa, to cause through their toxins dilatation of the blood-vessels of the mucous membranes and escape of blood-plasma, but particularly of colorless blood-corpuscles. *Acute gonorrhea in the male* commences in the most anterior portion of the urethra, particularly the navicular fossa being involved with especial severity, and only gradually the morbid process extends backward. Not rarely it stops before the compressor of the urethra, so that it then remains confined to the bulbous portion of the urethra. In contradistinction from this, *chronic gonorrhea in the male* is especially situated in the posterior portion of the urethra (membranous and prostatic portions). The mucous membrane in this situation is frequently thickened and covered with small papillary elevations—so-called *granular urethritis*. Here and there erosions and more profound losses of tissue occur upon the mucous membrane of the urethra—*ulcerative urethritis*; and if these undergo cicatrization, *stricture of the urethra* readily results, behind which a chronic inflammation may persist with especial obstinacy, although stricture of the urethra may develop, in the absence of previous ulceration, from contraction of hyperplastic submucosa.

Treatment.—No *specific remedy* in the treatment of gonorrhea is known. **Abortive treatment** of *acute gonorrheal urethritis in the male* has been attempted, and for this purpose especially injections of strong solutions of silver nitrate (1 : 30) have been employed, in order to cause exfoliation of the superficial layers of the urethral mucous membrane and to destroy the contained gonococci. This treatment can be serviceable only so long as gonococci are present alone in the superficial layers of the mucous membrane, therefore on an average within the first three days after the urethral discharge has made its appearance. A positive result is even then not always brought about, and, besides, inflammation of adjacent structures (urocystitis, epididymitis, etc.) readily takes place.

Men who are desirous of speedily getting rid of an attack of acute gonorrhea should remain in **bed** and receive a light **diet.** Strong coffee or tea, alcohol, strong acids, and condiments should be avoided. Boiled milk is especially advantageous. Daily **evacuation of the bowels** should be secured, if necessary by the use of stewed fruit (apple-sauce, stewed prunes) or of mild laxatives. Internally, **balsamics** are much administered, particularly the balsam of copaiba, although expectation should not be too sanguine :

℞ Balsam of copaiba, 0.6 (9 minims).
Make 20 such gelatin-capsules.
Dose: 1 capsule every two hours.

Urethral injections are largely employed, and for this purpose all sorts of *astringents* and *disinfectants* have been used. Among the many remedies that I have tried at my clinic a combination of *zinc sulphate* and *iodoform* has proved most reliable :

℞ Solution of zinc sulphate, 0.5 : 200 (7½ grains : 6½ fluidounces);
Iodoform, 5.0 (75 grains).—M.
To be well shaken and injected every two hours.

Urethral injections should be practised after previous micturition. A well-constructed glass *syringe*, which the patient can purchase under the less suggestive name of an ear-syringe, is employed for the purpose. It should be especially noted that the tip of the syringe is free from sharp angles and edges. The injection should be begun only after the syringe has been held vertically and all air has been expelled by carefully pushing the piston forward. The tip of the syringe is then introduced carefully but as deeply as possible into the urethra, while the fingers of the left hand keep the lips of the urethra firmly applied to the syringe, in order to prevent premature return of the fluid injected. The injection should be made quite slowly in order that the fluid may enter deeply into the urethra. At the completion of the injection the patient should keep the orifice of the urethra closed for an additional five or ten minutes; only in this way is it possible to maintain the irrigating effect of the fluid upon the urethral mucous membrane for some time. The method of injection is of great importance in the success of the operation. Among *astringent injections* many prefer silver nitrate (0.1–0.3 : 100), and recently protargol; others employ solutions of tannic acid (0.1–0.5 : 100), copper sulphate (0.1–0.5 : 100), lead acetate (0.5–1.0 : 100), etc. Among the *disinfectants*, carbolic acid (1.0–2.0 : 100), mercuric chlorid (0.005 : 100) and potassium permanganate may be mentioned.

Urethral injections should be persisted in so long as the discharge continues. It is even advisable to continue injections also for one or two weeks after the cessation of the discharge, but it will then be sufficient to repeat them thrice daily. Should the injections be suspended too early, the discharge frequently reappears. The patient will do well to abstain from sexual intercourse for a long time. The return to the use of alcoholic beverages should be made with great care. The physician should never fail to caution a gonorrheal patient against rubbing his eyes with his fingers without previous careful washing, as gonorrheal pus may be readily transferred, with the development of serious disease of the eyes.

Most gonorrheal patients will not obey the injunction of the physician to remain in bed, but will persist in continuing their work without interruption. The treatment under such circumstances will be the same, but the patients should wear a well-adjusted suspensory bandage and avoid physical exertion as much as possible, as otherwise epididymitis may readily develop as a complication.

Only brief reference will be made to the *treatment of the complications of gonorrhea.* When active *erections* and *chorda penis* occur the patient should take his evening-meal early, and this should consist exclusively of fluid. Before retiring the following powder should be taken:

℞ Sodium bromid,	2.0 (30 grains);
Lupulin,	0.5 (7½ “);
Camphor,	0.01 (⅛ grain);
Extract of licorice,	0.5 (7½ grains).—**M.**

Make 3 such powders.
Dose: 1 powder to be taken before retiring.

In bed the dorsal decubitus should be avoided.

In the treatment of *balanoposthitis* irrigation with tepid *carbolic-acid solution* (2.0 : 100) should be practised thrice daily and be followed by applications of *lead-water.* For this purpose absorbent cotton is best employed, being dipped in the solutions named. *Phimosis* and *paraphimosis* should first be treated with affusions of *lead-water;* but should these fail, resort may be had to *operation.* If *lymphangitis* has developed, affusions of tepid *lead-water* should be made; if suppuration result, the accumulations of pus should be evacuated with the *knife.* On the occurrence of *epididymitis* injections should at once be suspended. The affected epididymis should be elevated by means of a small pillow or of a cloth placed across the thighs. In addition, affusions of tepid *lead-water* should be made continuously to the epididymis. Daily evacuation of the bowels should be secured. Severe pain will often be relieved by the application of from 6 to 10 leeches to the perineum. I rarely apply a *dressing* to the testicle, and then employ moistened gauze-bandages. Should indurations in the epididymis persist, *potassium iodid* (5.0 : 200 ; 15 c.c.—1 tablespoonful —thrice daily) and inunctions of *ointment of potassium iodid* may be employed, although the results will generally be doubtful.

In the presence of *inflammation of the prostate gland*, the *seminal vesicles*, of *Cowper's glands*, of the *bladder* and the *kidneys* also the urethral injections should be suspended. In so far as the diseased structures are accessible *hot cataplasms* should be employed. If abscesses develop, they should be evacuated with the knife. When *cystitis* is present *salol* (1.0—15 grains—every two hours) should be administered and be followed by *oil of turpentine* (15 drops in milk thrice daily) in from ten to fourteen days. Should the inflammation become chronic, *irrigation of the bladder* will have to be resorted to.

Gonorrheal arthritis is most resistant to treatment. Among internal remedies I have obtained the best results from salol (1.0—15 grains—every two hours). Pain and inflammation are often mitigated by the application of an *ice-bag* to the affected joints. *Massage* with an ointment of potassium iodid, iodoform, or salicylic acid, or applications of tincture of iodin may also be tried. Recently, I have observed good results from *ligation of the member with a rubber tube*. Should suppuration occur, *surgical treatment* will be necessary. Other complications of acute gonorrhea should be treated in the usual manner.

The treatment of *chronic gonorrhea in the male* will vary in accordance with the anatomic alterations. In the treatment of the granular and ulcerative varieties of the disease *injections* have also been tried, the disinfectants and astringents already mentioned being employed, with a preference for the stronger solutions. It should, however, be clearly borne in mind that the materials injected are scarcely capable of reaching the posterior portion of the urethra, because the compressor of the urethra generally constitutes an insurmountable barrier. *Irrigation of the urethra* has, therefore, been undertaken, catheter-like instruments being introduced into the bladder, then being slowly withdrawn into the urethra and astringents and disinfectants being permitted to flow from a syringe connected with the catheter, particularly into the posterior portion of the urethra.

Treatment with sounds is also much practised ; metallic sounds with grooves at their anterior extremity into which astringent (tannic acid, silver nitrate) or disinfectant (iodoform) ointments are introduced especially being used. *Suppositories* of glycerin or cocoa-butter are also employed, mixed with the astringents and the disinfectants previously mentioned, introduced into the urethra and permitted to lie there until they melt. The treatment of chronic gonorrhea with *insufflations of disinfectants or astringents* is not much followed. If a stricture of the urethra be dependent upon gonorrhea, the former should be dilated by long-continued treatment with *bougies* or *dilators;* a cure cannot otherwise be expected. In addition, the treatment given for chronic gonorrhea may yet be necessary.

In the treatment of *acute and chronic gonorrhea in the female* the vagina should be carefully irrigated morning and evening with a *solution of mercuric chlorid* (0.1 : 1000), and then there should be introduced deeply into the vagina cotton-tampons with an *ointment of alum and iodoform* smeared upon their outer surface:

℞ Alum,	
Iodoform,	each, 10.0 (2½ drams);
Wool-fat,	
Lard,	each, 50.0 (1½ ounces).—M.
Apply topically.	

Surgical measures are often necessary, as, for instance, dilatation of the neck of the uterus and curetting of the cavity of the uterus. In the presence of complicating *Bartholinitis* cataplasms should be applied first, and as soon as fluctuation is evident the inflammatory focus should be incised with the knife. Should chronic suppuration persist, the gland must occasionally be enucleated with the knife.

The most certain *prophylactic measure against gonorrhea* consists in the avoidance of illegitimate sexual intercourse. The suggestion to evacuate the urine after intercourse and to make injections of dilute carbolic acid (2.0 : 100) into the urethra cannot with certainty be relied upon, and besides cannot always be carried out. The wearing of a guard over the penis or a *condom* affords protection only if it does not rupture. Houses of prostitution should be subjected to rigid medical supervision. An arrangement is said to exist in Belgian brothels according to which visitors are first examined medically before they are admitted. In order to remain unrecognized they are provided with a mask before being brought into the presence of the physician. The most dangerous practice consists in municipal prohibition of houses of prostitution, with their tacit tolerance. Young men suffering from gonorrhea should not be permitted to marry until they are wholly cured of their disease.

SOFT CHANCRE (CHANCROID).

Etiology.—The soft chancre consists of an ulcer whose purulent secretion is characterized by the intensity of its infectiveness. The *infective agents* are believed to be the bacilli first described by

Ducray and Krefting, which at times are grouped together consecutively in chains—so-called streptobacilli—and are characterized morphologically by the presence of a constriction at the middle, giving them the shape of dumb-bells. When stained by Gram's method they yield up the gentian-violet with which they have been stained. In addition to these bacilli pyogenic streptococci and staphylococci besides can generally be cultivated from the secretion from the ulcer.

In the majority of cases a soft chancre is acquired through *sexual intercourse* with a person suffering from soft chancre. The pus often penetrates into fissures and slight wounds of the sexual organs produced during the sexual act. It appears, however, also to happen that the pus may penetrate into the skin through the hair-follicles of the uninjured skin and give rise to chancroidal ulceration. Public women are generally the source of infection, and even in houses of prostitution under medical supervision a single infected prostitute, if the intervals between medical examinations are too long, is capable of infecting one hundred or two hundred men in the course of one or two weeks.

The circumstances mentioned explain why the majority of patients are *unmarried young men and women*. The occurrence of chancroid in *children* is attributable either to rape or to accidental extragenital infection. Physicians, midwives, and nurses are exposed to the risk of *extragenital infection* if with wounded fingers they are brought in intimate contact with patients suffering from chancroids and contaminate their wounds with chancroidal pus. The transference of chancroidal pus by means of *surgical instruments* seldom occurs at the present day, because it is customary to disinfect thoroughly all instruments before and after being used. Chancroids are occasionally transmitted also through ordinary *domestic utensils* (glasses, spoons, pipes) if chancroidal pus, as, for instance, in the presence of chancroid of the lips, has remained adherent to them, and which may be conveyed to healthy persons if the objects are transferred immediately from one person to another, in the same way as if articles contaminated with chancroidal pus are disinfected insufficiently. At times infection takes place through linen, clothing, or water-closets soiled with chancroidal pus. Sleeping with a patient suffering from chancroid is not unattended with danger. Soft chancre, like gonorrhea, is a purely *local disorder*. Recovery from one attack does not confer immunity from subsequent attack. Chancroid occurs not rarely *in association with other venereal disease*, particularly with gonorrhea, in the same person.

Symptoms.—Inoculation-experiments with chancroidal pus upon human beings have shown that the *period of incubation* is a short one. Within a few hours a reddened spot, and soon also a nodule—papule—will have developed at the point of inoculation.

In the course of twenty-four hours the overlying epidermis becomes raised into a vesicle. In the course of twenty-four hours more the epidermic covering ruptures, and a crust forms as a result of desiccation of the secretion. On removal of the latter an ulcer with all of the characters of a chancroid comes into view. Further, the discharge from a soft chancre is not infective at all periods; it loses its infectivity when the ulcer is about to undergo cicatrization. The soft chancre presents a distinctive **appearance.** It is generally circular in outline, and this undergoes considerable variation only if adjacent ulcers become confluent, or if the ulceration occurs along folds of the skin, when it assumes the form of fissures. The ulcer is sharply circumscribed, the margins appearing slightly elevated and feeling somewhat indurated. The margins descend steeply to the floor of the ulcer—crater-like. The floor of the ulcer appears excavated, almost worm-eaten, and is covered with purulent secretion. If the ulcer be grasped between the fingers, it bleeds readily, and the patient is conscious of pain. Frequently not one, but several ulcers are present—*multiple chancroids.* The ulcers vary in size between the head of a pin and a pea and more.

Left to itself, a chancroid undergoes gradual **cicatrization** in the course of five or six weeks. The secretion of pus becomes less, vigorous granulations form upon the floor of the ulcer, and finally a smooth, reddish cicatrix develops, which gradually becomes bright white and is surrounded only by a brownish ring of discoloration. After a time, however, every trace of the cicatrix will have disappeared.

Chancroids occur most frequently upon the *genitalia.* In the male they are especially common upon the inner surface of the prepuce, in the coronary sulcus of the glans, on the frenum of the prepuce, and upon the external border of the prepuce. Occasionally chancroids are situated upon opposed surfaces, obviously in consequence of auto-infection. The scrotum also is not rarely covered with chancroids. These are rarely situated within the urethra—concealed chancroid. Under such circumstances they give rise to a purulent discharge, which may be readily mistaken for gonorrhea. In women chancroids often develop on the inner surface of the labia, upon the posterior commissure, and upon the neck of the uterus. Chancroids may, however, occur upon any portion of the skin to which chancroidal pus is conveyed. Thus, it has been observed at the *anus* and in the *rectum,* either following unnatural sexual intercourse or as a result of inoculation with fingers contaminated with chancroidal pus, or in women possibly also from the overflow of chancroidal pus upon the perineum. Occasionally chancroidal ulceration occurs above the *symphysis pubis* or at the *umbilicus. Chancroids of the lips* have also been observed as a result of kissing or the use of pipes, drinking utensils, etc. Physicians and midwives occasionally acquire *chancroids upon the fingers*, and the like.

Among the *anomalous varieties of chancroid* the *follicular chancroid* may first be mentioned. This is characterized by its small size but great depth, obviously because the destructive process has taken place within a hair-follicle. The *superficial chancroid* offers a certain contrast, exhibiting a slight tendency to extend deeply, but tending rather to invade the neighborhood. The designation *bullous chancroid* is applied to the lesion presenting vesicular elevation of the skin, while the *luxuriating* or *elevated chancroid* in the stage of healing gives rise to the formation of unusually exuberant granulations. Occasionally the floor of a chancroidal ulcer exhibits a membranous, bacon-like deposit—*diphtheric chancroid;* or there may be blackish discoloration and gangrene of tissue—*gangrenous chancroid.* The gangrenous process may extend for a considerable distance and result in destruction of the penis and extensive ulceration of the cutaneous covering of the abdomen. The *phagedenic chancroid* is marked by a tendency for the ulcerative destruction to extend progressively in the absence of gangrenous alterations. Under such circumstances not only the cutaneous covering of the genitalia, but occasionally also that of the entire abdomen is destroyed, and the abdominal muscles may be exposed to view as if they had been dissected with the knife of a skilled anatomist. The *serpiginous chancroid* also is attended with progressive extension of the process of ulcerative destruction, while at other parts of the ulcer processes of cicatrization and healing are taking place.

Among the *complications* of chancroid *acute inflammation of the lymphatic glands—acute bubo*—is, on account of its frequency, deserving of mention first. The occurrence of this complication is favored by the irritation mechanically of the ulcer by trousers that are unduly tight, or by too much walking, or chemically by treatment that is too stimulating. Generally a single gland or a few glands become inflamed, and if the chancroid be situated on the genitalia the inguinal glands will suffer. The inflammation occurs upon the same side as that on which the ulcer is situated, because the phlogogenic agents are conveyed to the glands through the lymphatics; only in the presence of ulcers upon the frenum of the prepuce and in the middle of the penis may bilateral enlargement of the lymphatic glands occur, as the lymphatic vessels from both sides of the body meet in this situation. The exciting agents of the inflammatory process are in some cases pyogenic cocci—Streptococcus pyogenes, Staphylococcus pyogenes; but in other instances the specific bacteria of soft chancre are the active agents, and accordingly a distinction has been made between *non-specific* and *specific chancroidal buboes.* From the clinical point of view the two varieties are identical; a distinction can be made only

In the course of twenty-four hours the overlying epidermis becomes raised into a vesicle. In the course of twenty-four hours more the epidermic covering ruptures, and a crust forms as a result of desiccation of the secretion. On removal of the latter an ulcer with all of the characters of a chancroid comes into view. Further, the discharge from a soft chancre is not infective at all periods; it loses its infectivity when the ulcer is about to undergo cicatrization. The soft chancre presents a distinctive **appearance**. It is generally circular in outline, and this undergoes considerable variation only if adjacent ulcers become confluent, or if the ulceration occurs along folds of the skin, when it assumes the form of fissures. The ulcer is sharply circumscribed, the margins appearing slightly elevated and feeling somewhat indurated. The margins descend steeply to the floor of the ulcer—crater-like. The floor of the ulcer appears excavated, almost worm-eaten, and is covered with purulent secretion. If the ulcer be grasped between the fingers, it bleeds readily, and the patient is conscious of pain. Frequently not one, but several ulcers are present—*multiple chancroids*. The ulcers vary in size between the head of a pin and a pea and more.

Left to itself, a chancroid undergoes gradual **cicatrization** in the course of five or six weeks. The secretion of pus becomes less, vigorous granulations form upon the floor of the ulcer, and finally a smooth, reddish cicatrix develops, which gradually becomes bright white and is surrounded only by a brownish ring of discoloration. After a time, however, every trace of the cicatrix will have disappeared.

Chancroids occur most frequently upon the *genitalia*. In the male they are especially common upon the inner surface of the prepuce, in the coronary sulcus of the glans, on the frenum of the prepuce, and upon the external border of the prepuce. Occasionally chancroids are situated upon opposed surfaces, obviously in consequence of auto-infection. The scrotum also is not rarely covered with chancroids. These are rarely situated within the urethra—concealed chancroid. Under such circumstances they give rise to a purulent discharge, which may be readily mistaken for gonorrhea. In women chancroids often develop on the inner surface of the labia, upon the posterior commissure, and upon the neck of the uterus. Chancroids may, however, occur upon any portion of the skin to which chancroidal pus is conveyed. Thus, it has been observed at the *anus* and in the *rectum*, either following unnatural sexual intercourse or as a result of inoculation with fingers contaminated with chancroidal pus, or in women possibly also from the overflow of chancroidal pus upon the perineum. Occasionally chancroidal ulceration occurs above the *symphysis pubis* or at the *umbilicus*. *Chancroids of the lips* have also been observed as a result of kissing or the use of pipes, drinking uten-

[illegible] [illegible]. Physicians and midwives occasionally acquire chancroids [illegible] [illegible] fingers, and the like.

Among the anomalous varieties of chancroid the follicular chancroid must first be mentioned. This is characterized by its small size and great depth, obviously because the destructive process [illegible] place within a hair-follicle. The superficial chancroid [illegible] in contrast, exhibiting a slight tendency to extend [illegible] [illegible] [illegible] rather to invade the neighborhood. The [illegible] [illegible] chancroid is applied to the lesion presenting [illegible] [illegible] of the skin, while the luxuriating or elevated [illegible] [illegible] in the stage of healing gives rise to the formation of [illegible] exuberant granulations. Occasionally the floor of a [illegible] ulcer exhibits a membranous, bacon-like deposit—[illegible] [illegible]; or there may be blackish discoloration and gangrene of tissue—gangrenous chancroid. The gangrenous process may extend for a considerable distance and result in destruction of the penis and extensive ulceration of the cutaneous covering of the abdomen. The phagedenic chancroid is marked by a tendency in the ulcerative destruction to extend progressively in the absence of gangrenous alterations. Under such circumstances not only the cutaneous covering of the genitalia, but occasionally also that of the entire abdomen is destroyed, and the abdominal muscles may be exposed to view as if they had been dissected with the knife of a skilled anatomist. The serpiginous chancroid also is attended with progressive extension of the process of ulcerative destruction, while at other parts of the ulcer processes of cicatrization and healing are taking place.

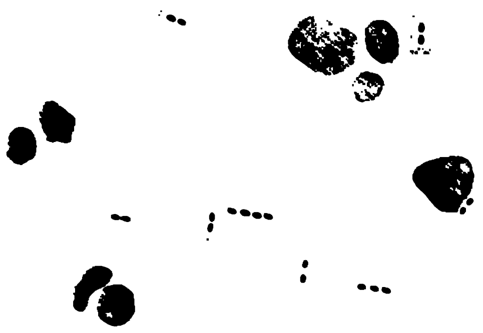
Among the complications of chancroid acute inflammation of the lymphatic glands—acute bubo—is, on account of its frequency, deserving of mention first. The occurrence of this complication is favored by the irritation mechanically of the ulcer by trousers that are unduly tight, or by too much walking, or chemically by treatment that is too stimulating. Generally a single gland or a few glands become inflamed, and if the chancroid [illegible] [illegible] on the [illegible] the inguinal glands will suffer. [illegible] [illegible] occurs upon the same side as that on which [illegible] [illegible], because the pyogenic agents are [illegible] [illegible] [illegible] the [illegible] only in the presence [illegible] [illegible] of the [illegible] and in the middle [illegible] [illegible] largement of the lymphatic glands [illegible] [illegible] [illegible] [illegible] of the body [illegible] [illegible] [illegible] agents of the inflammatory process [illegible] pyogenic [illegible]—[illegible] pyogenes. [illegible] [illegible] but in [illegible] the specific bacteria [illegible] the active agents, and [illegible] a distinction [illegible] between non-specific and specific chancroidal bubo [illegible] [illegible] point of view the two varieties are identical [illegible] [illegible] made only [illegible]ing should be applied. Occasionally [illegible] [illegible] are enlarged and inflamed without [illegible] [illegible] at all, and it will be necessary [illegible] [illegible] means. Other complications [illegible] [illegible] the usual rules.

[illegible] corresponds with that applicable [illegible] [illegible]rhea. Persons with chancroids should [illegible] [illegible] course until complete recovery has [illegible] [illegible] infecting others, and they should be [illegible] [illegible] menacing the public health if they [illegible] [illegible].

[illegible]IOUS DISEASES ATTENDED [illegible] [illegible] ALTERATIONS IN THE NERV[illegible] [illegible]

[illegible]MIC CEREBROSPINAL MENINGITIS.

[illegible] Epidemic cerebrospinal meningitis is one of [illegible] [illegible]ous *diseases*. Its exciting agent is believed [illegible] [illegible]coccus intracellularis discovered by Weichselbaum



[illegible] from the pus in a case of [illegible] [illegible]spinal meningitis; [illegible] methylene-blue; oil-immersion [illegible] [illegible] times (personal [illegible]

[illegible] the pneumoniacoccus, and, like this, is provided [illegible] Fig. 73). It is distinctive to find the micro- [illegible] pairs or more, within round-cells, whence the [illegible] intracellular.

In addition to meningococci, pyogenic cocci, streptococci, and staphylococci, also, are not rarely present in the meningeal exudate, and some clinicians assume that possibly the meningococci are not the sole exciting agents of the disease.

Epidemics of cerebrospinal meningitis are generally confined to *small areas*, as, for instance, frequently to a single barracks or isolated small communities and a few streets. Experience has shown that *children* are susceptible to the disease in an especially marked degree. Infection is favored by injuries of the skull, particularly by concussions, possibly, also, occasionally by emotional disturbances and excessive mental activity. Experience has shown that epidemics of meningitis occur mostly in the *cold season of the year*. Occasionally they persist for only a few weeks, but at times in isolated instances for many months, and even longer than a year. Further, *isolated—sporadic—cases* occur now and then.

With regard to the *mode of infection*, nothing of a definite nature is known. Personal intercourse, air, a third person, and inanimate objects appear to be capable of conveying the infection. Attention has been directed to the fact that the infective agents frequently appear to gain access to the cerebral meninges through the nasal cavities and their blood-vessels and lymphatics, and meningococci have often been found in the purulent nasal secretion. When epidemics of meningitis are prevalent meningitis not rarely complicates other infectious diseases, especially fibrinous pneumonia.

Symptoms.—The period of incubation of epidemic cerebrospinal meningitis is believed to be generally from three to four days, although under some circumstances it may be only a few hours. *Prodromes* are wanting, or manifest themselves in an indefinite manner for one or two days. In typical cases the disease begins with a single **chill**, which is followed by elevation of temperature to 39° or 40° C. (102.2° or 104° F.), and rarely above. The **pulse**, as compared with the temperature, is frequently slow, and this has been attributed to irritation of the vagus in consequence of the meningitis. **Nervous symptoms** soon make their appearance. The patients complain especially of intolerable headache, which is at times referred to the vertex, at other times to the temples or the occiput, but often is uniformly distributed over the entire head. It is not rarely attended with pulsation, and is greatly intensified by palpation and gentle percussion. Marked **hyperesthesia** to all sensory stimuli becomes manifest. Bright light and noises annoy the patient in an unusually marked degree, and slight pinching of the skin is felt as severe pain. Also increased irritability of the cutaneous vasomotor nerves is manifested by the appearance of red marks that persist for a long time on mechanical irritation of the skin, as, for instance, after passing a hard object over it—**meningitic spots.** **Consciousness** becomes progressively more obtunded, and the patients lie in a state of somnolence, are frequently delirious, and not rarely pass into a state of total unconsciousness, from which they awaken now and then with a loud cry—**hydrocephalic cry.**

One of the most important symptoms of cerebrospinal meningitis is **stiffness of the neck,** from the presence of which the disease has been designated also *rigidity of the neck* and *spasm of the neck.* Even on ocular inspection it will be noted that the patient lies with the head strongly flexed backward, so that the occiput appears deeply buried in the pillow. If an attempt be made to flex the head forward by means of the hand placed beneath it, insurmountable resistance is encountered, and the entire body moves in unison with the head. On the other hand, active flexion of the head posteriorly and rotatory movements can be executed without difficulty and pain. Obviously the stiffness of the neck depends upon contracture of the muscles of the neck, and this in turn is due to irritation of the cervical nerves in consequence of the meningitic process. Toward the close of life the rigidity of the neck not rarely diminishes, with progressive loss of consciousness, or it disappears entirely. The **vertebral column** acquires in the course of the disease a progressively increasing curvature forward—*opisthotonos*—so that the body is supported almost solely upon the occiput and the sacrum, and a fist can be introduced between the vertebral column and the bed. Often the vertebral column is extremely sensitive to pressure—*rachialgia.*

Often *symptoms* of **motor irritation and paralysis** are present. Thus, *inequality of the pupils* is frequently encountered; the pupillary reaction to light-stimulation may also be greatly diminished or wanting. Now and again spasm occurs in the muscles of mastication, and *grinding of the teeth* takes place periodically. While the *pulse* is at first slow, in consequence of irritation of the vagus, excessive acceleration occurs subsequently in consequence of paralysis of the vagus. The patients often suffer from copious **vomiting,** which also is dependent upon irritative processes in the path of the vagus. The abdomen often is scaphoid, or trough-shaped, so that occasionally the pulsation of the abdominal aorta can be seen beneath the abdominal wall. At the same time the abdominal walls are tense and hard, so that it seems probable that the condition is dependent upon contracture of the abdominal muscles. Also the muscular layer of the intestines appears to be in a state of unyielding contraction, so that the patients suffer from **constipation.** Not constantly, but frequently, it has been noticed that the **urine,** in spite of the presence of fever, is light in color, and voided in large amounts, and this has been attributed to vasomotor derangement. The urine not rarely contains albumin, occasionally, also, sugar. In most cases death takes place at the end of the first, or during the second, week of the disease, amid symptoms of excessive cerebral pressure, in consequence of paralysis of the brain or the heart. Occasionally, however, the disease is protracted for from four to six weeks, and even longer, with repeated remissions and exacerbations.

Among the *anomalies of epidemic cerebrospinal meningitis*, especially *abortive, fulminant,* and *apoplectiform cases* should be mentioned. The *abortive cases* are attended in times of epidemic only with headache, vertigo, and vomiting. These symptoms occasionally have disappeared in the course of one or two days; at times, it is true, especially if the patients do not take care of themselves, the condition passes into serious, fully developed meningitis. *Fulminating meningitis* may set in suddenly during perfect health, in the midst of work. The patient occasionally loses consciousness, and death may take place in the course of a few hours. It is noteworthy that upon post-mortem examination only the commencement of inflammation of the cerebral meninges is occasionally found. *Apoplectiform meningitis* is attended with sudden loss of consciousness and unilateral paralysis, so that the clinical picture resembles that of cerebral hemorrhage.

The designation *intermittent meningitis* has been applied to cases in which fever and other symptoms undergo exacerbation at definite and regularly recurrent intervals. This variety has nothing to do with malaria. It is dependent upon inflammation of the membranes with periodic exacerbations.

Among the *complications optic neuritis* and *choked disc* may be mentioned; these are important in diagnosis, and result from extension of the inflammatory process and from increased cerebral pressure. At times *paralysis of the ocular muscles* appears upon one or both sides. Occasionally *chemosis of the conjunctiva* and *protrusion of the eyeballs* develop, without thrombosis of the cavernous sinus. In some cases *ulceration of the cornea, purulent iridochoroiditis,* and *panophthalmitis* occur. *Roaring in the ears, impairment of hearing,* and *deafness* are dependent upon extension of the inflammatory process in the course of the auditory nerve to the labyrinth. Occasionally some of the extremities become the seat of *spasm* or *paralysis* in unilateral or in crossed distribution. The *knee-jerk* is at times increased, at times absent, at times unaltered. Not rarely *facial herpes* is observed. Occasionally symptoms of *polyarthritis* appear, and this may be attended with suppuration in the joints, in the pus from which meningococci have been found.

Among the *sequelæ chronic hydrocephalus* may be mentioned, and which occasionally may rather suddenly terminate fatally after the lapse of months. In some cases blindness, deafness, paralysis, or contractures may persist. The patients complain with especial frequency for a long time of headache, vertigo, and impairment of memory, which may appear even after slight physical or mental activity.

Diagnosis.—The diagnosis of epidemic cerebrospinal meningitis is not difficult. The disorder is distinguished from **non-epidemic purulent meningitis** by the fact that it occurs as an independent disease, apart from other infectious disease, traumatism, or adjacent inflammation. *Bacteriologic diagnosis* also has been resorted to, cerebrospinal fluid obtained by lumbar puncture being examined for the presence of meningococci. The fluid itself is almost always flocculent, even turbid and purulent, rarely clear. Epidemic meningitis is distinguished from **tuberculous meningitis** by the fact that the latter is much more insidious in onset, progresses more slowly, and with slighter fever; that it generally occurs in tuberculous patients, and is not rarely attended with the development of tubercles in the choroid, which can be recognized with the ophthalmoscope as yellowish, circular dots. It is also of diagnostic importance that in cases of tuberculous meningitis clear cerebrospinal fluid is almost always obtained on lumbar puncture, and which in the course of twenty-four hours precipitates a veil-like coagulum. The presence of tubercle-bacilli in the fluid is decisive in the diagnosis. If labial herpes be present, it is indicative rather of epidemic than of tuberculous meningitis.

Prognosis.—The prognosis of epidemic cerebrospinal meningitis is **serious**, as most cases terminate fatally; but even those in which life is preserved are frequently attended with irremediable damage, to which reference has already been made in the consideration of the sequelæ.

Anatomic Alterations.—On removal of the bony calvarium the **diploe of the cranial bones** will be conspicuous on account of the presence of a large amount of blood. The **dura mater** is greatly stretched, and dry upon its inner surface. After removal of the dura mater the surface of the brain will be conspicuous on account of its **expanded gyri** and **shallow sulci**, as a result of which an impression is gained as if the brain had been compressed from within toward the inner surface of the skull. The **blood-vessels of the pia and the cerebral cortex** are greatly distended with blood, and punctate and striate, greenish-yellow, purulent infiltrations especially are noticeable in the pia in the course of the blood-vessels. In advanced cases adjacent collections of pus have coalesced and enclose the convexity of the brain in an almost complete purulent capsule. Toward the base of the brain the formation of pus generally diminishes. The columns of the arachnoid also are infiltrated with pus, so that the anatomic alterations consist in purulent arachnitis and piitis—thus a purulent leptomeningitis. On opening the **ventricles of the brain**, these will be found greatly dilated. They are filled with an excessive amount of ventricular fluid, characterized by flocculent or purulent turbidity. Occasionally the **choroid plexus** is the seat of purulent infection. Corresponding alterations are encountered also in the **pia and the arachnoid of the spinal cord.** They are generally most pronounced upon the posterior aspect of the cord, probably as a result of the dorsal decubitus persistently maintained by the patient. The lesions are slightest in the cervical portion of the spinal cord, which occasionally appears almost free from disease.

On *microscopic examination*, in addition to dilatation of the blood-vessels abundant inflammation of the pia and arachnoid with round-cells will be found. Here and there the same process extends into the substance of the brain and the spinal cord through the processes of the pia, and in these situations small collections of pus may have formed. Meningococci can often be demonstrated in the round cells.

Treatment.—Persons suffering from epidemic cerebrospinal meningitis must at once go to **bed**, even if the disease be of the mildest variety. Whenever possible the **sick-room** should be kept quiet and slightly darkened. In any event, the head of the patient should be placed toward the window, in order to prevent the glaring effect of the sunlight upon the eyes. The **food** should be wholly liquid, and consist principally of boiled and susequently cooled milk. Ice-bags should be applied to the head, on either side of the neck, and along the vertebral column, and should be promptly replaced by others as soon as the ice has melted. **Phenacetin, lactophenin,** or **antipyrin** should be employed for the relief of fever and headache:

℞ Phenacetin,	1.0 (15 grains).

Make 10 such powders.
Dose: 1 powder thrice daily.

℞ Lactophenin,	0.5 (7½ grains);
Sugar,	0.3 (4½ ").—M.

Make 10 such powders.
Dose: 1 powder thrice daily.

℞ Antipyrin,	1.0 (15 grains);
Sugar,	0.3 (4½ ")—M.

Make 10 such powders.
Dose: 1 powder thrice daily.

Should headache be considerable, resort must be had to regularly repeated **injections of morphin**:

℞ Morphin hydrochlorate,	0.3 (4½ grains);
Glycerin,	
Distilled water,	each, 5.0 (75 minims).—M.

Dose: 8 minims, subcutaneously, twice or thrice daily.

Occasionally the pains subside after cerebrospinal fluid has been evacuated by **lumbar puncture**, and the intracranial pressure is thereby diminished. It may also happen that the patient is again **restored** to consciousness. It is true the improvement generally **is only transitory**, as the cerebrospinal fluid usually reaccumulates in increased amount. Generally little will be accomplished with **derivatives** and **antiphlogistics**, as, for instance, with leeches applied to the temples or the mastoid process, with inunctions of mercurial ointment, potassium-iodid ointment, or iodoform-ointment. Recently **hot baths** at a temperature of 40° C. (104° F.), and of ten minutes' duration, have been recommended as an admirable remedy; some personal experiences, it is true, have not been favorable. For *prophylactic purposes* it is wise to isolate patients with epidemic cerebrospinal meningitis, and to disinfect their clothing and the rooms occupied by them. If a case occurs in a house with numerous inmates, the healthy individuals should be placed in other rooms.

SECONDARY PURULENT CEREBROSPINAL MENINGITIS.

Secondary purulent meningitis agrees with epidemic cerebrospinal meningitis with regard to its anatomic alterations and clinical manifestations. From the etiologic standpoint, however, the former is distinguished from the latter by the fact that it does not occur epidemically, and that it does not represent an independent affection, but always develops as a complication of some other antecedent disease. As an example of such disease, **extension of inflammatory processes** is, in the first place, to be taken into consideration. Disease of the petrous process of the temporal bone and of the internal ear, of the nose, and the frontal sinuses, the cerebral sinuses, and the brain-tissue—abscess—gives rise to purulent meningitis with especial frequency. Also, erysipelas of the face and scalp, and phlegmons and furuncles of the same parts are occasionally followed by purulent meningitis. Purulent meningitis is not rarely of **traumatic origin.** Under such circumstances open wounds of the skull especially are likely to be operative, but occasionally purulent meningitis has been observed to develop after violent concussion of the entire body. At times purulent meningitis develops in the sequence of **infectious diseases.** Thus, it has been observed in association with fibrinous pneumonia, typhoid fever, smallpox, measles, scarlet fever, articular rheumatism, gonorrhea, and septicopyemia.

Various bacteria may take part in the development of purulent meningitis. In some cases the Meningococcus intracellularis, the causative factor of epidemic cerebrospinal meningitis, is the active agent; in others, however, ordinary pyogenic cocci (streptococcus, staphylococcus), and in still others specific bacteria, as, for instance, typhoid-bacilli in cases of typhoid fever, or the pneumococcus of Fraenkel in cases of fibrinous pneumonia. The Bacterium coli also has, in a number of instances, been found in the [illegible] meningeal pus, apparently indicating infection from the intetinal tract. Occasionally the bacteriologic diagnosis can be made during life if lumbar puncture is practised, and the fluid obtained is examined for bacteria.

The **diagnosis** is not rarely attended with difficulty from the fact that the disease develops most insidiously, and then escapes recognition.

The **prognosis** and the **treatment** are the same as those for epidemic cerebrospinal meningitis (pp. 447 and 448).

SEROUS CEREBROSPINAL MENINGITIS.

The soft membranes of the brain exhibit an unmistakable tendency to respond to inflammatory irritation with suppurative inflammation. Serous meningitis occurs but seldom. Occasionally it sets in *acutely*, and then in its course resembles at times rather a purulent, at other times a tuberculous, meningitis; while in other instances it pursues a *chronic course*. In the latter event alcoholism acts as the cause. The acute varieties of the disease probably develop as a result of the activity of *bacteria* and their toxins, and the pneumoniacocci of Fraenkel and colon-bacilli have been demonstrated in the serous exudate. This disease formerly was generally designated cerebral edema—acute and chronic hydrocephalus—and its inflammatory nature was not recognized. The acute cases are distinguished from purulent meningitis by the fact that the fluid obtained on lumbar puncture is not turbid but clear, and in contradistinction from tuberculous meningitis the fluid does not contain tubercle-bacilli. The fluid is distinguished from normal cerebrospinal fluid by its higher specific gravity, the presence of a larger amount of albumin, and the greater pressure to which it is exposed. Occasionally, lumbar puncture is followed not merely by amelioration of the symptoms, but even by recovery. The *prognosis* is, accordingly, more favorable than that of purulent and tuberculous meningitis, but it is nevertheless quite serious.

TETANUS.

Etiology.—The causative agent of tetanus is known and was shown by Flügge and Nicolaier (1884) to be the **tetanus-bacillus.** This organism occurs frequently and in large number in horse-manure, and it has been found also in garden-earth, dust, and cobwebs. Its growth in pure culture is difficult, and it can be successfully inoculated in animals, as, for instance, mice. In pure culture the microörganism often exhibits a drumstick-like enlargement, corresponding to the situation of a spore (Fig. 74).

Tetanus-bacilli most frequently gain entrance into the human body through wounds, so that most cases of tetanus are of *traumatic* origin. Tetanus, like erysipelas, is, therefore, a *traumatic* *infectious disease.* Whether a wound be large or small is wholly a matter of indifference with regard to the occurrence of the disease; it is only necessary that the wound be accidentally infected with tetanus-bacilli, and this may happen the more readily if it be not at once thoroughly disinfected with solutions of carbolic acid (from 3 to 5 per cent.) or of mercuric chlorid (0.1 per cent.), and if, after disinfection has been completed, it is not protected from subsequent infection by a protective bandage. Tetanus may appear in the newborn child if tetanus-bacilli gain entrance into the umbilical wound. At times tetanus occurs in recently delivered women if infection of the placental site in the uterus takes place. In cases of traumatic tetanus tetanus-bacilli will be found always only in the wound itself, and not distributed throughout the entire body; and it must, therefore, be assumed that toxins of the tetanus-bacilli gain entrance into the general circulation, and exert their effects upon the central nervous system, especially upon the spinal cord and the medulla oblongata. It has been ascertained that the toxicity of the toxins of the tetanus-bacilli is extraordinarily great, and upon this fact depends the circumstance that occasionally the symptoms of tetanus follow rapidly after wounds of the foot.

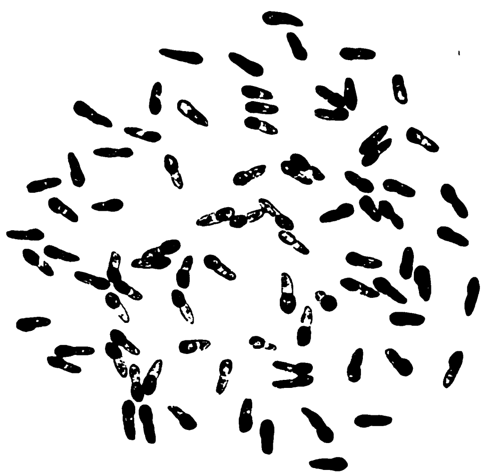


FIG. 74.—Spore-containing tetanus-bacilli, from a pure culture; carbol-fuchsin preparation; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

The occurrence of tetanus is favored by certain conditions or *contributory factors*. One of these is *mental depression*. In times of war tetanus has been observed more frequently among the vanquished than among the victors. Etiologic significance is attached also to *temperature-influences*, as numerous cases of tetanus have occasionally been observed in the late summer, when sultry days are followed by unusually cool nights. *Wounds of the nerves* also are especially feared by surgeons, as well as *contused and lacerated wounds*, and *wounds in which splinters are lodged*, as well as *wounds of the fingers and toes*. Tetanus occurs also with greater frequency in overcrowded, poorly ventilated, and especially filthy *hospital-wards*. Certain *racial distinctions* also have been said to exist. Negroes especially are believed to suffer more frequently from tetanus than whites, but possibly the greater want of cleanliness on the part of the negroes may explain the suppositious racial distinction.

Traumatic tetanus has further been differentiated from *rheumatic* and *idiopathic tetanus*, exposure to cold being made responsible for the former, while no cause whatever has been demonstrable for the latter. At the present day both varieties are advantageously included in the designation *cryptogenetic tetanus*. In many cases even of this kind the condition may be one of traumatic tetanus, in which by reason of its smallness the wound is not noticed by the patient, and meanwhile has healed completely. It is possible also that infection of internal wounds (as of the mucous membranes) may have taken place, and these naturally are not accessible to examination. At any rate, cases of cryptogenetic tetanus are the less common the more carefully wounds are looked for.

In former years tetanus occasionally occurred epidemically in surgical clinics and in maternities, and in some of the latter the majority of parturient women and newborn children at times died from tetanus. At the present day such conditions are no longer encountered, because most scrupulous attention is given to cleanliness, and to the isolation of patients suffering from tetanus.

Symptoms.—The *period of incubation of tetanus* probably is occasionally but a few hours, and this should not occasion surprise in view of the great toxicity of the tetanus-toxins. Nevertheless, tetanus may not follow a wound until the lapse of several days, because infection of the wound may take place only after the wound has been inflicted.

Abnormal sensations in the wound or the cicatrix, increased secretion of pus, and reopening of the cicatrix, as well as nervous restlessness and sleeplessness, have occasionally been mentioned as *prodromes*. The distinctive symptoms of tetanus consist in **tonic muscular spasm**, which is at times interrupted by more violent tonic, and especially clonic spasm. At the same time **reflex irritability** is abnormally increased. The earliest symptoms consist in stiffness and impaired mobility of the muscles. Occasionally the *muscles of mastication* are involved first, so that the act of chewing is rendered difficult, and soon becomes wholly impossible. The teeth are firmly clenched, so that the patient speaks through them, and on attempts to depress the lower jaw forcibly with the fingers a sense of unyielding resistance is encountered. On palpation the masseter muscles are found to be tense and as hard as a board, and if the face be emaciated the outlines of the muscles named stand out prominently beneath the skin. This constitutes the well-known symptom of *lock-jaw* or *trismus*. In the newborn tetanus is occasionally confined essentially to lock-jaw, so that the condition has been designated also trismus of the newborn. The remaining *muscles of the face* also take part in the tonic contracture. The buccal orifice appears increased in width and slightly open, so that generally the teeth are visible between the lips. As a result the lower portion of the face acquires a laughing expression—the so-called sardonic grin (p. 453, Fig. 75). The nasolabial folds are deep, and the nasal alæ appear drawn outward. The palpebral fissure is narrowed, and the upper portion of the face presents a tired and sleepy expression. The forehead is often thrown into deep transverse folds, as if the patient were taken by astonishment and surprise. The facial expression in a case of tetanus therefore portrays various emotions, which together constitute the characteristics of the *tetanic facies*. Occasionally *strabismus* occurs.

The patient often complains of *spasm of the pharynx*, which greatly interferes with the taking of food, and may render it wholly impossible. Tonic contracture of the *muscles of the back* gives rise to abnormal curvature of the vertebral column. Most commonly the spinal column forms an arch with its convexity forward—*opisthotonos*—so that the body rests upon the bed only with the occiput and the sacrum, and the fist can be readily introduced between the spine and the bed. Less commonly there is marked convex curvature posteriorly—emprosthotonos—or toward one side—pleurothotonos—or the body is stretched out straight—orthotonos. The whole body may be lifted from the occiput like a stiff rod.

Tonic spasm of the *thoracic muscles* will, in the first place, give rise to marked interference with the respiratory movements, and, besides, the contracted muscles not rarely can be felt with the hands to be as hard as a board, and their outlines may even be visible beneath the skin. Tonic contraction of the *abdominal muscles* would give rise to retraction and board-like hardness of the abdominal walls. The *muscles of the extremities* also are generally involved in tonic contracture, at times in extension, and at other times in flexion, and accordingly they may offer considerable resistance to passive movement.

From time to time the muscular contractures are intensified paroxysmally, and are temporarily converted into clonic spasm. This change apparently occurs spontaneously, but especially as a result of reflex irritation. Even loud sounds, a bright light, a draft of air, and, still more, pressure and touch are capable of inducing *tetanic attacks*. **Consciousness** generally is preserved in cases of tetanus, and delirium occurs but rarely. **Cutaneous sensibility,** as a rule, also exhibits no alteration. The **tendon-reflexes,** which often cannot be carefully tested in consequence of the

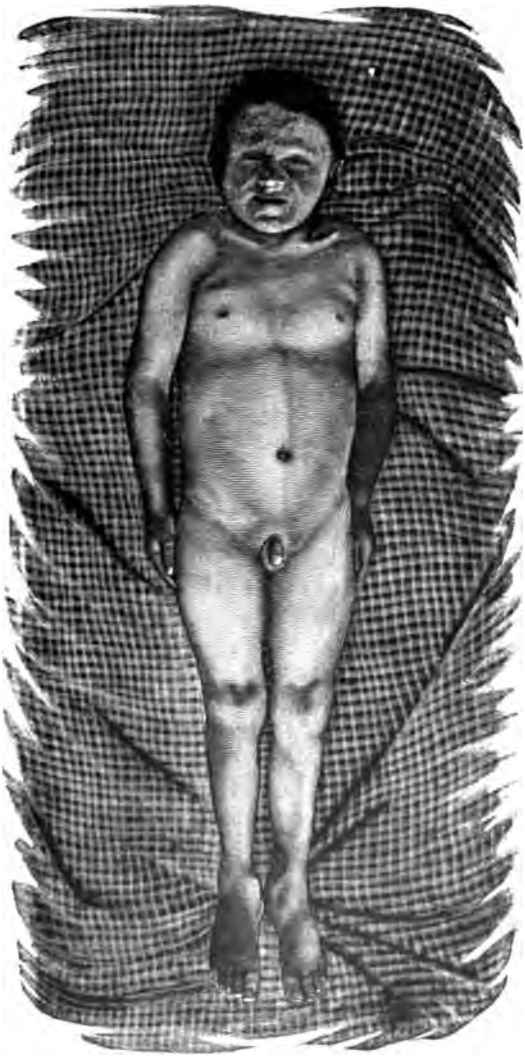


Fig. 75.—Tetanic facies in a case of traumatic tetanus in a boy 13 years old: **from a photograph** (personal observation, Zurich clinic).

muscular contracture, exhibit no peculiarity. The **bodily temperature** is often unchanged; occasionally, however, elevation of temperature to 43° C. (109.4° F.), and above—**hyperpyrexia**—has been observed, and this is in all probability scarcely dependent upon the muscular spasm, but rather upon innervational disturbances in the nerve-centers controlling the bodily temperature. The **pulse** remains regular, and is but temporarily less full, or not at all palpable, during the paroxysm of tetanus, because the strongly contracted muscles and the tense tendons interfere with its palpability. Occasionally derangement in **micturition** and **priapism** occur, in consequence of tetanus of the muscles of the penis. The urine not rarely contains albumin.

Tetanus may pursue an acute, a subacute, or a chronic *course*. Some cases terminate fatally within a few hours. Chronic cases are occasionally protracted over several weeks, and frequently exhibit remissions and exacerbations. Death generally occurs in a paroxysm of tetanus, in consequence of cerebral or cardiac paralysis.

Among the *anomalies of tetanus* is *localized tetanus*, in which the tetanic manifestations are confined to one or more members or to trismus, or occur in unilateral distribution. Cephalic or hydrophobic tetanus also is worthy of mention. This generally develops in connection with wounds of the face, and is attended with facial paralysis, frequently with simultaneous contracture of the facial muscles upon the same side and spasm of the pharynx on any attempt at swallowing.

Diagnosis.—The diagnosis of tetanus is easy in view of the readily recognized symptoms. From **meningitis**, which also may be attended with contracture of the muscles of the nucha and rigidity of the neck, tetanus is distinguished, among other things, by the fact that it is unattended with headache and progressive loss of consciousness, while these are conspicuous in cases of meningitis.

Prognosis.—Tetanus is an exceedingly grave disease. The longer the affection is protracted the more likely, it is true, is recovery to take place. Elevation of temperature and delirium are indications of unfavorable significance.

Anatomic Alterations.—Distinctive anatomic alterations in cases of tetanus are as yet unknown. Not rarely hemorrhage takes place upon and into the meninges, as well as into the brain and the spinal cord, although these are insignificant secondary changes and results of the circulatory disturbances induced by the muscular spasm. Recently fine structural alterations in the ganglion-cells of the spinal cord have been described, although it has not been definitely demonstrated that these findings are peculiar.

Treatment.—Patients suffering from tetanus in surgical cs must be isolated, because conveyance of the disease through bandages and articles of linen, through instruments and unclean nurses' hands, is not impossible. The **sick-room** should be as quiet as possible, and be capable of being rendered dark. **Milk** is to be recommended as the diet, and under some circumstances it may be administered at regular intervals by means of a tube introduced through the nares into the stomach. **Wounds** should be carefully inspected for the presence of foreign bodies, and should be enlarged if secretion has collected within them. The wounds should then be disinfected and bandaged. Occasionally resort has been had to amputation of members. Recently **specific treatment of tetanus** with **tetanus-serum** has been attempted by Behring, but the results have as yet been neither convincing nor brilliant, because generally the treatment was begun too late. The serum is prepared in the dye-works of Meister Lucius at Höchst, and can be obtained from that source.[1] The results of treatment with a powdered **tetanus-antitoxin** prepared by Tizzoni, which can be preserved more conveniently, and for a longer time than the serum, and which is dissolved in water before being used, have not been more brilliant. Both preparations possess antitoxic, but not bactericidal, properties; that is, they do not destroy tetanus-bacilli that may be present, but only neutralize the toxins generated by them. Injections of cerebrospinal tissue also have been employed recently, this tissue combining with the tetanus-toxin and rendering it innocuous.[2]

Many clinicians confine themselves to **symptomatic treatment**, directing their efforts principally to reducing the abnormally increased reflex irritability of the spinal cord. For this purpose *bromids* and *chloral hydrate* especially are to be recommended, as, for instance:

℞ Solution of sodium bromid,	30.0 : 200	(1 ounce : 6½ fluidounces);
Potassium bromid,	15.0	(½ ");
Ammonium bromid,	5.0	(75 grains).—M.

Dose: 15 c.c. (1 tablespoonful) thrice daily.

℞ Solution of chloral hydrate, 5.0 : 200 (75 grains : 6½ fluidounces).
Dose: One-half to be used as an enema morning and evening.

Occasionally delirium occurs after the use of chloral hydrate, but it is without especial significance. The employment of *opium* and *morphin* is less to be recommended than that of chloral hydrate, because these agents rather increase the reflex irritability of the spinal cord, and in frogs even induce tetanic muscular spasm. *Curare* also is not a serviceable remedy, as it has no influence upon the central nervous system, and only inhibits the motility of the muscles without in any way influencing the tetanic state of the spinal cord. Some clinicians have observed good results from application of the *galvanic current to the spinal column.*

---

[1] Antitetanic serum has been marketed by a number of reliable manufacturers. —A. A. E.

[2] Successful results have been reported from the treatment of tetanus with subcutaneous injections of carbolic acid (2 per cent.) in doses of 15 minims given intervals of two or three hours.—A. A. E.

Preventive measures—prophylaxis—are of great importance, as with all traumatic infectious diseases. Every wound, however slight, should be disinfected with 5 per cent. solution of carbolic acid, or 0.1 per cent. solution of mercuric chlorid, and then be protected from subsequent contamination by a bandage. Cobwebs, an old hemostatic in domestic use, should not be applied to open wounds. Special consideration should be given to contused wounds and to impacted splinters of wood, as infection with tetanus-bacilli may readily take place under such circumstances.

INFECTIOUS DISEASES OF VARYING LOCALIZATION.

I. DIPHTHERIA.

PHARYNGEAL DIPHTHERIA.

Etiology.—The exciting agent of all diphtheric diseases, as also of pharyngeal diphtheria, consists in the **diphtheria-bacilli**, first thoroughly studied by Löffler in 1884.

Diphtheria-bacilli are small rods with rounded extremities, frequently with a bulbous enlargement at one end, for the staining of which Löffler's methylene-blue especially is to be recommended. They can be stained also by the method of Gram. In stained preparations it will be seen that especially the two poles of the diphtheria-bacilli are colored deeply, while the central portion of the body is stained irregularly, and therefore appears filled with colorless granules (Fig. 76). Diphtheria-bacilli can be readily cultivated upon solidified blood-serum from the calf or the sheep. Toxins have been separated from them, which in part contain albumin, and are therefore so-called *toxalbumins*.

Although infection with diphtheria-bacilli is the principal requirement for the development of pharyngeal diphtheria, the condition is generally not one of pure infection with diphtheria-bacilli, but is a *mixed infection* with other bacteria, particularly with the **Streptococcus pyogenes**, and it is not without reason assumed that the principal dangers of pharyngeal diphtheria are attributable to the streptococcic infection.

Pharyngeal diphtheria is preëminently a *disease of childhood*, but cases appear by no means rarely in adults. The disease occurs t times *sporadically*, at other times *epidemically*. Infection takes ce occasionally through *immediate contact* with the sick, as, for ıce, in nursing, and especially through kissing, or the disease be conveyed through a *third person* or *inanimate objects* (clothing, letters, books, articles of food, particularly milk). Dissemination of the etiologic agent through the *air* is possible also for a short distance. As with the majority of infectious diseases of childhood, so also with pharyngeal diphtheria, *schools* and *public playgrounds* especially are the media through which children most frequently acquire their infection, in consequence of association with other sick children or with members of the family.

Infection may be favored by certain circumstances—*contributory factors*. These include *exposure to cold* and *misty, damp weather, and climate*.

There are *diphtheria-houses* and *diphtheria-apartments*, whose infectivity is not always due to the fact that after the previous occurrence of diphtheria disinfection of the rooms was not effected with sufficient care, if at all. I knew of such a house in Göttingen, in which extensive and thorough disinfection failed to eradicate the disease, while no further cases of diphtheria occurred in the house after a drain in the basement was emptied and walled up. Sometimes the occurrence of pharyngeal diphtheria is favored by *antecedent infectious disease*, as, for instance, scarlet fever; although it must be stated explicitly that most inflammations of the pharynx in the course of infectious diseases, although they closely resemble diphtheria, are due to other bacteria, with especial frequency the Streptococcus pyogenes alone.

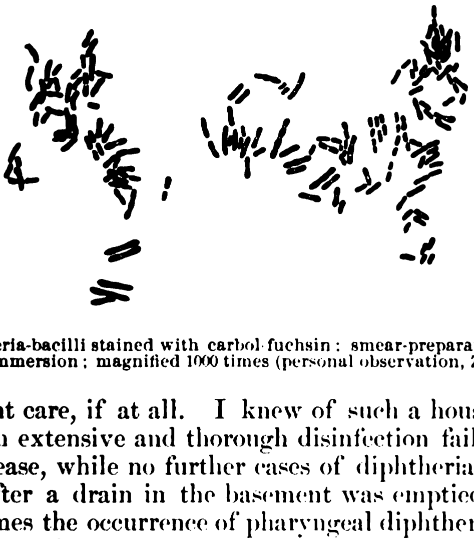


FIG. 76.—Diphtheria-bacilli stained with carbol-fuchsin: smear-preparation from a pure culture; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

In some families an *hereditary* or *congenital tendency—predisposition—to pharyngeal diphtheria* is unmistakably to be recognized, although it is not possible to demonstrate especial peculiarities in the anatomic structure of the tonsils, which are considered as the portal of entry for the diphtheria-bacilli. Individuals with *enlarged—hyperplastic—tonsils* are attacked by diphtheria with especial frequency. Recovery from pharyngeal diphtheria confers no immunity for the future, but, on the contrary, an increased *pre-*

disposition to repeated attack is observable. Infants during the first six months of life generally remain exempt from pharyngeal diphtheria. The majority of attacks occur *between the second and the seventh year*, while the number of cases diminishes again after the fourteenth year.

Until the middle of the nineteenth century pharyngeal diphtheria was a rare, an almost unknown, infectious disease in certain professional quarters. In recent years the distribution, and especially the fatality, of the much-feared disease are thought to have diminished, although an explanation for this circumstance is wanting.

Symptoms.—The *period of incubation of pharyngeal diphtheria* is generally from two to seven days, although in some cases it appears to be but a few hours. Prodromes do not occur constantly, but are quite common, consisting especially in a feeling of general malaise. Occasionally the local manifestations are preceded for a short time by general symptoms. In other cases, however, the latter only follow the pharyngeal manifestations. Chilliness, even a chill, and **elevation of temperature** to 39° C. (102.2° F.) and above not rarely inaugurate the train of symptoms. Soon, however, patients complain of **pain in the pharynx**, which is increased especially on speaking and swallowing, and also on rotatory movements of the head. The neck and the head, therefore, are frequently directed toward the side and held stiffly. Examination beneath the angle of the jaw will generally disclose enlargement of the **submaxillary lymphatic glands**—consensual bubo—upon one or both sides, which are tender on pressure. Not rarely, in consequence of augmented secretion of mucus, **efforts at swallowing** are increased, and these may be attended with pain. Speech often has a **nasal clang**, if the tonsils are considerably enlarged and reduce the nasopharyngeal space, and in breathing the mouth is not rarely kept open.

Inspection of the pharynx discloses varying appearances, in accordance with the severity of the disease. In some cases a **diphtheric catarrh** will be found, which, like every other form of catarrh, is attended with unusual redness and swelling of the mucous membrane and with increased secretion of mucus. At times this condition is confined to circumscribed portions of the pharyngeal structures, as, for instance, the tonsil and the arch of the palate on one side, while at other times it involves all of the pharyngeal structures. Not rarely **lacunar pharyngeal diphtheria** is observed, involving principally one or both tonsils, and being attended with a collection of yellowish or grayish deposits in the lacunæ of these structures, which are often surrounded by a zone of redness. The designation *necrotic pharyngeal diphtheria* is applied to cases in which, in consequence of necrosis, the diseased structures are themselves transformed into a pulpy, grayish, occasionally blackish mass. The highest grade of this condition consists in *gangrenous pharyngeal diphtheria*, which has been designated also *septic pharyngeal diphtheria*, because it is frequently attended with general septicemia. Patients presenting this variety of pharyngeal diphtheria generally give off an offensive odor from the mouth. The pharynx is the seat of extensive gangrenous lesions, which at times give rise to marked destruction and dangerous hemorrhage. *Fibrinous—croupous—pharyngeal diphtheria* is a rare variety, which is attended with the deposition of a fibrinous exudate upon the surface, particularly of the tonsils, and which has formed a fibrinous membrane that can be detached from the surface of the tonsils without noteworthy loss of tissue.

Gangrenous or septic pharyngeal diphtheria especially is justly to be feared, because it is likely to be attended with profound general septic infection. It is often wholly unattended with fever. In fact, in some patients the bodily temperature is subnormal. Generally, profound pallor of the skin and the mucous membranes develops quickly as a result of rapid diminution in the number of red blood-corpuscles and in the percentage of hemoglobin, with simultaneous increase in the number of white blood-corpuscles—hyperleukocytosis. In the general appearance of the patient progressive apathy and loss of strength are manifest in marked degree. The pulse becomes smaller and smaller, frequently also irregular and slow. The area of cardiac dulness becomes increased in consequence of dilatation, especially of the right side, and it generally extends beyond the right sternal line, while the heart-sounds become faint and masked and sometimes are scarcely audible. Death not rarely occurs suddenly from cardiac paralysis, particularly on assuming the erect posture.

The *duration of pharyngeal diphtheria* varies from a few days to two weeks. The disease but rarely persists for a longer time. Among the *complications* inflammatory processes resulting by extension and by metastasis are to be distinguished. Not rarely diphtheric alterations extend to adjacent mucous membranes, and thereby augment the dangers of the disease.

Diphtheria of the nasal mucous membrane is frequently first disclosed by *occlusion of the nares*. *Nasal discharge* takes place, which at first is generally serous, but subsequently becomes bloody and putrid, and which often so irritates the skin of the upper lip, over which it flows, that this becomes reddened and swollen in places. Not rarely necrotic shreds are expelled in blowing the nose, having been exfoliated from the inflamed nasal mucous membrane. Grayish-green, necrotic masses of tissue can frequently also be recognized from the external nasal orifices. When nasal diphtheria occurs as a complication the prognosis becomes exceedingly grave, as the majority of patients die as the result of general septicemia and exhaustion.

Extension of pharyngeal diphtheria to the laryngeal mucous membrane also is of grave significance, and gives rise to symptoms of *laryngeal diphtheria—laryngeal croup.* The patients are principally children, in whom the symptoms assume the character of acute laryngeal stenosis, which often terminates fatally from asphyxia. Should pharyngeal diphtheria extend to the *Eustachian tube* and the *mucous membrane of the internal ear*, the patient will complain of severe pain in the ear, and suppuration, rupture of the tympanic membrane, and other symptoms and dangers of inflammation of the middle ear may result. Occasionally extensive inflammation of the cervical connective tissue below the lower jaw and in the neck occurs, with the development of *Ludwig's angina.* The skin generally presents a pallid, alabaster-like appearance and feels hard and infiltrated. Pressure is painful. The movements of the jaw and the head are interfered with. Should suppuration ensue, the pus may rupture externally, but occasionally it burrows downward into the mediastinum or into the pleural or pericardial cavity; or the large vessels of the neck are eroded, and fatal hemorrhage occurs. Under all circumstances the phlegmons of the cervical connective tissue are among the serious complications.

Among the *metastatic inflammatory processes acute nephritis* is the most common, occurring in many cases, further, not as a complication, but rather as a sequel, and being apparently dependent less upon dissemination of bacteria than upon the elimination of diphtheric toxins. The danger from this condition consists not in uremia, which, as experience has shown, occurs but seldom in this connection, but rather in the circumstance that it may be transformed into the chronic variety of nephritis, and finally, as a result, may terminate fatally.

Occasionally painful articular swellings occur in the course of pharyngeal diphtheria—polyarthritis; but inflammatory processes may develop in any organ, as, for instance, pneumonia, pleuritis, endocarditis, pericarditis, etc. In some cases *changes in the skin* appear in the course of pharyngeal diphtheria, as, for instance, roseolous or erythematous exanthems or cutaneous hemorrhages. At times *dissemination of diphtheria-bacilli to other portions of the body* may be effected through the patient himself, as, for instance, to external wounds. Infection of the eye is especially serious.

Among the *sequelæ of pharyngeal diphtheria affections of the nerves* especially are deserving of mention. Often *post-diphtheric peripheral paralysis* occurs. The muscles of the palate and the pharynx are paralyzed with especial frequency. Speech acquires a nasal character in consequence of defective closure of the naso-pharyngeal space, and foreign material may enter the larynx in eating and drinking, so that food ingested passes from the pharynx into the nose, and in part escapes externally through the nasal orifices. If the arch of the palate and uvula be touched with a hard body, they fail to exhibit contraction. Under such circumstances there is, additionally, danger that ingested matters may enter the larynx and then find their way further downward, and, becoming lodged in the smallest bronchi and pulmonary alveoli, give rise to foreign-body pneumonia or aspiration-pneumonia, or to abscess and gangrene of the lung. Naturally, the dangers last named are augmented if pharyngeal diphtheria is additionally complicated by *paralysis of the muscles of the larynx.* Paralysis of the thyro-aryepiglottic muscle especially is to be considered in this connection, as it is the important function of this muscle to close the entrance to the larynx during the act of deglutition by drawing the epiglottis backward. Further, post-diphtheric paralysis of the laryngeal muscles may also occur independently of paralysis of the muscles of the palate.

At times *neuritis* or *polyneuritis* develops in the nerves of the extremities. Under such circumstance I have observed also ascending polyneuritis, the nerves and the muscles of the extremities, then those of the arms, being first paralyzed, and eventually, also, cerebral nerves being involved, when death resulted in consequence of paralysis of the vagus. Grave danger to life results from *paralysis of the phrenic nerve and of the diaphragm. Paralysis of accommodation* also occurs, and can be readily recognized from the fact that the patients are unable to read because they cannot clearly recognize the letters. Occasionally tabiform symptoms are present—*post-diphtheric pseudotabes.* The gait is ataxic, the knee-jerks are wanting, and when the eyes are closed marked swaying occurs. In addition, paresthesia and hyperesthesia, even anesthesia, of the soles of the feet and the legs are present. The symptoms are attributable to inflammation of the nerves of the legs, the cutaneous nerves especially being affected. Although *spinal and cerebral lesions* occur in the sequence of pharyngeal diphtheria, they are far less frequent than neuritic alterations.

Diagnosis.—The diagnosis of pharyngeal diphtheria can be made with certainty only by means of *bacteriologic examination* of the inflammatory products, for the same anatomic alterations in the pharyngeal mucous membrane are excited also by wholly different bacteria, with especial frequency by the Streptococcus pyogenes. By means of a platinum-loop a small amount of the inflammatory products is removed from the pharyngeal mucous membrane, and from this material cover-slip preparations and pure cultures are made. Those who are not sufficiently trained in such manipulations should send the material for examination to a bacteriologic institute. Careful bacteriologic examination is necessary, also, for the reason that streptococcic angina and anginas due to other bacteria generally pursue a more favorable course than pharyngeal diphtheria.

Prognosis.—The prognosis should be guarded in all cases pharyngeal diphtheria; even cases that are at first mild occasionally acquire a grave character in their further course. Gangrenous pharyngeal diphtheria is justly considered an especially grave disease on account of the great danger of general septicemia.

Anatomic Alterations.—On examination of necrotic areas in the pharyngeal mucous membrane diphtheria-bacilli will be found almost always only in the uppermost layers; they but rarely penetrate deeply, and are conveyed through the circulation to other viscera. In addition to diphtheria-bacilli the necrotic masses contain also other bacteria, particularly streptococci, which frequently penetrate into the deeper layers and thence into the general circulation. The necrotic portion is generally separated from the approximately healthy portions of tissue by a wall of round cells. The necrosis is in part due to the action of bacterial poisons (toxins) upon the tissues, but in part also to the fact that the exudate deposited in the mucous membrane contains an abundance of fibrin, the coagulation of which results in compression of the blood-vessels, with interruption of the circulation.

The **heart** is generally flabby and pale. On microscopic examination granular cloudiness, and fatty and vitreous degeneration are generally found in the muscular fibers of the myocardium, together with nuclear multiplication or degeneration. Foci of interstitial inflammation also occur frequently: round-cell accumulation, with compression-atrophy of the adjacent muscle-fibers, often only with pigmentary remains of muscle-fibers. Here and there collections of bacteria will be encountered. The **kidneys** likewise are pale and flabby. Microscopic examination discloses with particular frequency fatty degeneration and vacuolation in the epithelial cells of the convoluted uriniferous tubules. In addition, interstitial round-cell accumulations and hemorrhages occur not rarely, and occasionally, also, glomerulitis. Inflammatory alterations have, in a number of instances, been demonstrated in the **peripheral nerves.** Occasionally the inflamed nerves were thickened in places—*nodose neuritis.*

Treatment.—In the treatment of diphtheria it is believed that a *specific remedy* has been found in the **antidiphtheric serum** of Behring—*diphtheria-antitoxin*—but there are still a number of competent and experienced physicians who are not wholly convinced of the curative powers of the serum.[1] The remedy is injected subcutaneously (1000 immunity-units on the average), and in severe cases the injection is repeated on the succeeding days, according to circumstances. The serum contains certain substances—antitoxins—which, although they do not destroy the diphtheria-bacilli, neutralize the toxins produced by them. In Germany the serum employed is prepared under official supervision in the chemical works of Meister Lucius, in Höchst, and can be purchased in drug-stores; while in Switzerland a preparation manufactured in Berne is generally employed. Other countries, also, have established institutes for the preparation of diphtheria-antitoxin.[1] Unpleasant accidents of a serious nature seldom occur after injection of the serum, although occasionally erythematous or roseolous exanthems, muscular and articular pains, albuminuria, and even hematuria are observed [2]

---

[1] This opinion appears too conservative. Both the prophylactic and the curative value of diphtheria-antitoxin are admitted almost universally. Through the use of this remedy the mortality from diphtheria has been reduced one-half. It is important, however, to make the dosage adequate to the needs of the individual case. No serious secondary effects are to be feared if the serum be concentrated.—A. A. E.

In addition to injections of serum, attention should be given to thorough cleansing of the inflamed pharyngeal structures, and for this purpose I prefer **gargling** to **irrigation** of these parts with a spray-apparatus, because the latter is capable mechanically of irritating the inflamed structures. The process of cleansing should be undertaken especially after each meal, in order to prevent accumulation and decomposition of the remains of food. For irrigation *sodium salicylate* is to be recommended:

℞ Solution of sodium salicylate, 30.0 : 500 (1 ounce : 16 fluidounces).
For external use.

Potassium chlorate is to be preferred to all other drugs as a gargle:

℞ Solution of potassium chlorate, 5.0 : 200 (75 grains : 6½ fluidounces).
To be used as a gargle.

Great relief, and also a favorable effect upon the inflammatory alterations, will generally be yielded by the **swallowing of bits of ice,** and the application of an **ice-collar about the neck.**

The **diet** should be only fluid, with a preference for milk. If symptoms of general septicemia be present, extensive use of concentrated alcoholic preparations should be made. It is also not rarely necessary to increase the vigor of the heart by means of **stimulants,** as, for instance, by subcutaneous injections of camphorated oil.

After convalescence has set in, gargling of the pharyngeal cavity with a solution of salicylic acid or of potassium chlorate should be continued so long as diphtheria-bacilli can be found in the secretions. Children should be kept from school until the oropharyngeal cavity no longer contains diphtheria-bacilli, in order to prevent dissemination of the disease.

Among the *prophylactic measures systematic irrigation of the* geal cavity with a solution of mercuric chlorid (1:10,000) mentioned. This should be practised after each meal, rly at times when an epidemic of pharyngeal diphtheria In children with enlarged tonsils *tonsillotomy* should be d if they exhibit a tendency to tonsillitis. When pharyn- iphtheria occurs in a family it is advisable to place the t in a well-conducted hospital, and the contents of the sick- , together with the furniture, body-linen, and clothing, should isinfected. If the patient remain at home, the healthy chil- n should be removed to a place free from pharyngeal diphtheria, , as a precautionary measure, they should receive injections of m 300 to 500 immunity-units of Behring's *antidiphtheric serum*. nder all circumstances the healthy members of the family should e kept from school, as they are capable of conveying the infective matter, even though they themselves remain well. The *kissing* of diphtheria-patients should be strictly forbidden, because conveyance may readily take place by this means. During the prevalence of an extensive epidemic of pharyngeal diphtheria it is advisable to close the schools until the epidemic has been suppressed.

---

[1] In the United States diphtheria-antitoxin is prepared and marketed by reputable firms and also, for eleemosynary purposes principally, by boards of health. —A. A. E.

[2] These effects have been shown to be due principally to the large amounts of serum that it has been necessary to inject, and they are observed less frequently since concentrated sera have been prepared and used.—A. A. E.

LARYNGEAL DIPHTHERIA.

Etiology.—Laryngeal diphtheria, which is known also as *laryngeal croup*, is generally a **secondary disease** that is likely to occur as a complication of antecedent pharyngeal diphtheria. The occurrence of independent, or primary, diphtheria of the laryngeal mucous membrane is wholly denied by some physicians. My own experience, however, is in favor of the view that in rare instances *idiopathic diphtheria of the larynx* does occur. As may be understood, laryngeal diphtheria, like the pharyngeal diphtheria by which it is generally preceded, is preëminently a *disease of childhood* being observed most frequently *between the second and the seven year of life*. As in the case of pharyngeal diphtheria, laryng diphtheria occasionally occurs in the sequence of other *infecti diseases*, particularly measles, although in the majority of instan the conditions are similar only anatomically, while etiologic other bacteria, especially streptococci, are usually the respon agents.

Anatomic Alterations.—The anatomic alterations of geal diphtheria almost always consist in that **variety** of mation that it is customary to designate **croupous** or **fi** Under such circumstances a fibrinous exudate is poured o urface of the laryngeal mucous membrane, undergoi forming membranes up to several millimeter can readily be removed from t means of forceps witho ages **are freque**

upon the surface directed toward the laryngeal mucous membrane. The fibrinous inflammatory process often extends from the larynx to the trachea, and even into the bronchi. In the smaller bronchi the fibrinous exudate naturally loses its membranous character generally, and rather resembles inspissated pus. In the larynx itself, especially thick deposits accumulate upon the posterior surface of the epiglottis, and upon the true vocal bands, to the latter of which they generally adhere with especial tenacity.

On *microscopic examination* an intricately ramified network of fibrinous threads is found in the fibrinous membranes, in the midst of which colorless and here and there also red blood-corpuscles are found arranged in groups and nests. The epithelial cells of the laryngeal mucous membrane are often transformed into homogeneous plates, without nuclei and not stainable, and it has not without reason been assumed that this coagulation-necrosis of the epithelial cells renders possible the coagulation of the fibrinous exudate.

Laryngeal diphtheria is but rarely attended with *necrotic inflammation*, such as is especially peculiar to pharyngeal diphtheria. Nothing of a definite nature whatever is known with regard to *diphtheric catarrh of the larynx*. In addition to symptoms of laryngeal diphtheria, **changes in the lungs**, such as atelectasis, acute emphysema, and inflammation, are generally found in those dead of the disease.

Symptoms.—The symptoms of laryngeal diphtheria consist in **acute stenosis of the larynx**, frequently attaining a fatal intensity, and which is dependent upon closure of the chink of the glottis by fibrinous deposits upon the true vocal bands, with obstruction to the entrance of air. The disease often begins with **hoarseness**. To this cough is added, possessing a peculiar barking or stridulous character, so that it has been designated also **croupy cough**. Gradually symptoms of laryngeal stenosis appear. Inspiration becomes attended with a loud hissing, or sawing sound —**croupy stridor**—which may often be heard across several rooms. At the same time inspiration is slow and prolonged. Expiration is generally unobstructed and quiet. The **total number of respiratory movements** accordingly is generally diminished. Respiration is evidently embarrassed—**objective dyspnea**. Shortly before each inspiration—pre-inspiratory—the nasal alæ are dilated, and generally also the mouth is opened. The sternomastoid muscles become contracted during inspiration, and the larynx is drawn downward. The intercostal spaces become retracted during inspiration, and the ensiform cartilage and the epigastrium are generally so greatly retracted that the lower end of the sternum closely approximates the vertebral column.

All of the manifestations of objective dyspnea described are a natural result of the effort of the respiratory muscles to overcome the existing obstruction, while, besides, the lungs are not sufficiently filled with air and distended during inspiration, so that with the inspiratory dilatation of the chest the yielding portions of the thorax are forced inward by the external atmospheric pressure. The marked inspiratory contractions of the diaphragm finally result in retraction of the lower extremity of the sternum and the diaphragm. The greater the dyspnea the more marked is the **cyanosis**, in consequence of excessive accumulation of carbon dioxid and deficiency of oxygen in the blood. Naturally with these pallor of the skin is usually associated, so that the lips, the tongue, the cheeks, and the auricles acquire rather a leaden-gray or livid appearance. Speech is reduced to so faint a whisper that what is spoken is only with difficulty intelligible when the ear is held close to the mouth of the patient. The **facies** is expressive of great fear. The child becomes restless, throws itself about in bed, wishes at times to sit up or to be carried upon the arm; then, again, to be placed back in bed; and nowhere finds rest and amelioration. It is, therefore, not remarkable that even such patients as are quiet and good-natured in health become impatient and unmanageable.

The **bodily temperature** is frequently, though not constantly, elevated, and but rarely above 39° C. (102.2° F.). The **pulse** is accelerated, and is conspicuous for its smallness, often, also, for its irregularity. The **heart** frequently is dilated, particularly its right side, so that the right border of cardiac dulness extends beyond the right sternal border. The heart-sounds are running and generally faint. The **cervical veins** are occasionally converted into cords as thick as the little finger in consequence of venous stasis. The **excretion of urine** is often almost wholly suppressed. If urine be voided, it generally contains albumin, and often also tube-casts.

From time to time marked increase in the dyspnea occurs, so that the patient appears to be threatened with suffocation, and not rarely death takes place in such an *attack of croup.*

Attacks of this kind occur with especial frequency during the night, and in sleep. The patient is suddenly awakened from sleep, in desperation grasps the larynx at the site of obstruction to the air-current, cries, and states that he is suffocating, catches hold of the bed or the nurse, and breathes amid evidences of most profound obstruction to respiration. Occasionally attacks of croup subside quite rapidly when the patient has expectorated fibrinous membrane, which indicates that the attack was a result of the lodgment of croupous membrane above, and closure of, the chink of the glottis during sleep. At times accumulations of mucus, or adhesions between the free borders of the vocal bands, constitute the cause for attacks of croup: while, on the other hand, the older view that periodically occurring attacks of muscular spasm in the adductors of the vocal bands excited the attacks of croup is scarcely correct, as laryngoscopic examination shows that in the presence of laryngeal diphtheria the muscles of the vocal bands are rather in a paretic state, which, among other circumstances, is readily explained from the serous infiltration of the muscular tissue.

The *duration of laryngeal diphtheria* is not rarely from one to three days; occasionally the disease is protracted for a week. Only exceptionally does the disease last longer, although isolated instances have been recorded in which croupous membranes were expelled for more than nine weeks.

Death is generally the result of asphyxia, which may be caused by extreme laryngeal stenosis or by the supervention of lobular pneumonia, or both together.

Among the *complications of laryngeal diphtheria bronchitis* and *bronchopneumonia* are most frequently encountered. The latter is by some physicians designated croupous pneumonia in consideration of the primary disease. Occasionally the violent respiratory movements cause *rupture of pulmonary alveoli*, in consequence of which *interstitial emphysema of the lungs*, and, secondarily, *subcutaneous emphysema* (earliest in the jugular fossa), or occasionally also *pneumothorax* result. *Acute emphysema of the lower and median borders of the lungs* will be recognized from the fact that the upper border of liver-dulness is unusually low, and the area of cardiac dulness is diminished in extent. *Atelectatic areas in the lungs* must attain an extent and thickness of at least four or five centimeters in order to give rise to dulness on percussion and to bronchial breathing. In contradistinction from pneumonic foci, they are characterized by the fact that they disappear within a short time as soon as the airless portions of the lungs are again distended with air. *Pericarditis* and *endocarditis* are rare occurrences. The *liver* often is increased in size in consequence of venous hypostatic hyperemia. *Delirium* and *coma* occur when the blood contains an excess of carbon dioxid. Death is occasionally preceded for a short time by *clonic muscular contractions.*

Among the *sequelæ of laryngeal diphtheria* long-continued *hoarseness*, chronic *bronchitis*, and *pneumonia* may be mentioned. In some patients also distention of the lungs—that is, *alveolar emphysema of the lungs*—persists. At times laryngeal diphtheria is followed by *scrofulosis*, *pulmonary tuberculosis*, or, after some time, by miliary tuberculosis.

Diagnosis.—In the differential diagnosis of laryngeal diphtheria other varieties of acute laryngeal stenosis, such as occur in children, especially in consequence of marked catarrhal states, as **pseudocroup**, must first be taken into consideration; further, the entrance of **foreign bodies into the larynx** and the presence of **retropharyngeal abscess**. **Acute edema of the glottis** occurs but seldom in children. In cases of retropharyngeal abscess a fluctuating prominence will be visible and palpable upon the posterior pharyngeal wall, while in the presence of *foreign bodies* the history is generally important in the diagnosis. In the differentiation between *pseudocroup* and *croup* inquiry should be directed to determining whether diphtheric alterations are present in the pharynx, which would be in favor of croup. In addition, instructions should be given to collect all of the expectorated and vomited matters in order that they may be examined for croupous membrane. The fact that pseudocroup is generally unattended with danger to life, and that the symptoms of acute laryngeal stenosis do not, as in the case of croup, increase progressively, but frequently disappear during the day, to return on the subsequent night, as well as that pseudocroup not rarely is frequently repeated in children in the course of years, whereas croup generally occurs but once in children, can but seldom be depended upon in the differential diagnosis.

The most rapid and the most certain means of clearing up the diagnosis consists under all circumstances in *laryngoscopic examination*, the practice of which in children, it is true, is in and of itself difficult, but which is particularly attended with great difficulty in children in a state of threatening suffocation. In the laryngoscopic image the interior of the larynx will be seen to be covered with the characteristic fibrinous deposits. The vocal bands exhibit, besides, deficient respiratory and phonatory mobility.

Attention should be directed at this place to the fact that not all fibrinous inflammations of the laryngeal mucous membranes are caused by diphtheria-bacilli. The circumstance that diphtheric lesions are present in the pharynx would be in favor of laryngeal diphtheria. Should these be situated upon the posterior surface of the pharyngeal structures, they may readily escape detection.

In cases of idiopathic or primary laryngeal diphtheria the diagnosis can be made with certainty only when *diphtheria-bacilli* are demonstrated in the expectorated shreds of membrane. The same anatomic alterations are, in rare instances, induced by *other bacteria*, as for instance, streptococci; and even *toxic influences* (the accidental entrance into the larynx of ammonia or acids, the inhalation of the vapors of chlorin, burns of the skin) may bring about similar anatomic alterations.

Prognosis.—The prognosis of laryngeal diphtheria is most serious, as the majority of children die in consequence of asphyxia. The younger the child the greater is the danger, for especially in childhood, and particularly in early childhood, the laryngeal cavity is relatively quite small, so that even slight obstructions to the air-current may be followed by serious consequences. In addition, the results of tracheotomy, if rendered necessary, are the less certain the younger the child operated upon.

Treatment.—The treatment of laryngeal diphtheria resembles wholly that of pharyngeal diphtheria in all cases in which laryngeal diphtheria occurs as a complication of pharyngeal diphtheria. **Subcutaneous injections of Behring's antitoxic serum** have, however, also been recommended in the treatment of primary laryngeal diphtheria. In addition, in order to facilitate the expulsion of the croupous membrane, the **air of the room** should be kept moist by vaporizing hourly, by means of the inhalation-apparatus of Siegle, sodium-chlorid solution (0.5 per cent.) or lime-water, at a considerable elevation. Lime-water, lactic acid, and other solvents of fibrinous membranes have also been employed by **inhalation,** although success is scarcely to be expected on account of the dilution of the solutions and because the inhaled fluid scarcely penetrates to the deeper portions of the larynx.

Patients with laryngeal diphtheria should always be provided with experienced and intelligent **nurses,** who know at once what should be done in a case of croup, when suffocation is threatened. An **emetic** should be kept in readiness, for occasionally membranes are expelled from the larynx in the course of the act of vomiting, after which the danger from suffocation lessens; for instance:

℞ Solution of copper sulphate, 1.0 : 100 (15 grains : 3 fluidounces).
Dose: 10 c.c. (2½ fluidrams) to be taken every ten minutes until vomiting occurs.

℞ Solution of apomorphin hydrochlorate, 0.2 : 10 (3 grains : 2½ fluidrams).
Dose: 3 minims by subcutaneous injection.

The most certain means of averting the danger of suffocation either consists in the introduction through the mouth of metallic tubes into the pharynx, down to and between the vocal bands—**intubation of the larynx**; or the trachea is opened below the true vocal bands by means of an incision, into which a silver tube—**tracheal cannula**—is introduced—**tracheotomy.** As the relatives generally object to bloody operations upon the patient, intubation is at the present day practised earliest and more frequently than tracheotomy, although it sometimes happens that it does not afford the desired relief, and tracheotomy subsequently becomes necessary. In this connection it should be borne in mind that neither operation will any longer be capable of affording relief when the bronchi or the lungs are already involved in the inflammatory process, and the respiratory area is excessively contracted and diminished. It is, therefore, important not to undertake the performance of the operation too late. It may also be of advantage to employ injections of antitoxic serum after the operation.

The *prophylaxis of laryngeal diphtheria* is the same as that of pharyngeal diphtheria (p. 464).

NASAL DIPHTHERIA.

Etiology.—Nasal diphtheria is generally **a secondary disease,** complicating antecedent pharyngeal diphtheria. *Primary nasal diphtheria* occurs but seldom. From the nature of the causative factors it can be understood why the disorder is most common in *children.*

The **anatomic alterations** are generally those of a necrotic, less commonly those of a fibrinous, inflammation.

The **symptoms** are the same as those described on p. 459.

The **diagnosis** is not difficult, but should always be confirmed by bacteriologic methods.

The **prognosis** is grave, as death may result from septicemia.

The **treatment** consists in the employment of Behring's **antitoxic serum,** in systematic **irrigation of the nose** with solution of mercuric chlorid (0.1 per cent.) or with lime-water, or in **tamponade of the nares** by means of absorbent cotton through the anterior nasal orifices.

The diphtheria-bacillus is capable of thriving in other situations than upon the mucous membrane of the pharynx, the larynx, and the nose, although such disease belongs to other departments of medicine. Occasionally *diphtheria develops upon external wounds* or upon the *ocular conjunctiva.* Necrotic alterations in the mucous membrane of the esophagus, the stomach, the intestines, the biliary passages, and the urinary passages are likewise designated diphtheric by such clinicians as do not restrict the name diphtheria to those diseases that are induced by diphtheria-bacilli. Under such circumstances other bacteria are generally operative.

II. TUBERCULOSIS.

CHRONIC PULMONARY TUBERCULOSIS.

Etiology.—Chronic pulmonary tuberculosis is generally known as *pulmonary consumption* or *pulmonary phthisis,* and is an exceedingly widespread disease, as about one-seventh of all deaths are due to it.[1] Like all tuberculous diseases, chronic pulmonary tuberculosis also is dependent upon the **tubercle-bacillus** discovered by Koch in 1882. Nevertheless, it should be borne in mind that, in most cases, other bacteria, and among these **pyogenic cocci,** especially Streptococcus pyogenes, also are found, together with the tubercle-bacilli, so that in the majority of cases of pulmonary tuberculosis a *mixed infection* is present, in which the tubercle-bacilli, it is true, play the fundamental *role* under all circumstances.

---

[1] With a view to precision, the use of the terms "consumption" and "phthisis" in place of "tuberculosis" is to be discouraged, inasmuch as tuberculosis is not invariably attended with destruction and wasting, while, on the other hand, these may attend non-tuberculous processes. In addition, the terms objected to imply a more unfavorable prognosis than the more strictly correct term, and have a correspondingly more depressing effect upon the patient.—A. A. E.

Infection with tubercle-bacilli will occur the more readily if favored by *contributory factors.* Among these, in the first place, the *conformation of the body—constitution*—is to be included. Experience has shown that generally not thick-set and robust persons are attacked, but rather tall, delicate, pale persons, with a long, narrow, and imperfectly distensible chest, a bodily constitution to which the designation *phthisical habitus* also has been applied. This is transmitted by heredity in some families, so that the physical conformation is *hereditary.*

Infection with tubercle-bacilli is favored further by a *deficiency of pure, fresh air.* Persons who spend the entire day in closed factories and workrooms, and inhale *dust,* are not rarely attacked by pulmonary tuberculosis, while the disease is rare in mountaineers and in sailors. A distinct difference is appreciable between those who live in the country and those who live in the city, to the detriment of the latter; and among urban dwellers those are attacked with especial frequency that live in overpopulated, damp, manufacturing cities. It can be understood that the danger of infection is increased by all such conditions as diminish the resisting powers of the body, including *deficient nutrition, long-continued disease, anxiety, grief,* and *emotional disturbances* of all kinds. It is also comprehensible that *other antecedent disease of the lungs* will render these organs more susceptible to infection with tubercle-bacilli. *Injury to the chest,* without obvious external wound, also favors infection.

Two varieties of chronic pulmonary tuberculosis are to be distinguished—*primary* and *secondary.* In cases of primary pulmonary tuberculosis the lungs become independently the seat of disease, while in cases of secondary tuberculosis infection of the lungs is consequent upon tuberculosis of some other viscus.

Primary pulmonary tuberculosis probably is generally of *aërogenic origin*—that is, tubercle-bacilli are inhaled with the air. The air in the vicinity of persons suffering from pulmonary tuberculosis contains tubercle-bacilli, because in the act of coughing, of sneezing, and even of speaking, a portion of the sputum containing tubercle-bacilli is disseminated in the air in the form of minute drops. In addition, the sputum of tuberculous patients, if expectorated without special precautions, dries, and becomes converted into powder, inhalation of which likewise gives rise to infection. Constant association with patients suffering from pulmonary tuberculosis is accordingly not free from danger, and *sisters in religious orders* are, therefore, frequently attacked by that disease, as, in the first place, they are in nursing brought much in association with patients suffering therefrom, and, besides, they live a good deal indoors. Pulmonary tuberculosis is a frequent disease also in *prisons*. Prisoners are permitted to be in the open air but little, and several are confined together in small rooms. The wearing of *linen* and *articles of clothing* from cases of pulmonary tuberculosis has been shown to be not without danger of infection if the articles have not been thoroughly sterilized. The danger of infection with pulmonary tuberculosis is indicated by the fact that *husband and wife* not rarely infect each other. It is true that occasionally the one first affected has died several years previously before the other presents marked symptoms of pulmonary tuberculosis.

Formerly *hereditary transmission of pulmonary tuberculosis* was considered the most frequent mode of infection. That inheritance of pulmonary tuberculosis is possible is free from all doubt, for tuberculous, and even cavernous, lesions have been encountered in the lungs of the newborn; the number of instances on record, however, is but small, so that it may be assumed with certainty that hereditary pulmonary tuberculosis is one of the least common varieties. The experience that tuberculosis is widely distributed in some families is dependent not upon hereditary transmission of the tubercle-bacillus, but upon the fact that the phthisical habitus is inherited, and that the members of such families are from youth compelled to associate with tuberculous patients.

Alimentary pulmonary tuberculosis includes such cases as have resulted from the ingestion of articles of food containing tubercle-bacilli. In this connection especially milk, and, in lesser degree, meat from tuberculous cows, are to be taken into consideration. Tuberculosis in cattle is, unfortunately, a most widespread disease, and therefore the danger of infection is not slight. If, under the circumstances mentioned, it is more plausible to assume first tuberculous infection of the intestine, the possibility also is not to be excluded that tubercle-bacilli may pass through the lymphatic structures of the intestinal mucous membrane without inflicting injury, and may become lodged first in the lungs.

Infection of the lungs through *sexual intercourse* also is not to be omitted from consideration. Tubercle-bacilli have, in a number of instances been found in the seminal fluid of tuberculous men when the sexual organs were wholly free from tuberculous lesions. Likewise sexual intercourse with women suffering from tuberculosis of the urinary or generative organs is not unattended with danger for healthy men, because tubercle-bacilli are readily capable of entering the urethra, whence they may be further disseminated, and, among other organs, reach the lungs.

Secondary pulmonary tuberculosis occurs in conjunction with antecedent tuberculosis in other organs, and is, to a certain degree, *metastatic*—*embolic*—in character, tubercle-bacilli being conveyed through the lymphatics or the blood-vessels to the lungs. Any viscus the seat of tuberculosis may be the source of origin for pulmonary tuberculosis, namely, bones, joints, skin, lymphatic glands, intestines, larynx, and urogenital tract, etc. It appears noteworthy that not rarely tuberculosis of the tracheobronchial lymphatic glands precedes pulmonary tuberculosis. Inhaled tubercle-bacilli are capable of penetrating first the stomata of the pulmonary alveoli, and of passing thence through the lymphatics of the lungs, and they may be intercepted by the tracheobronchial lymphatic glands, in which tuberculous alterations may take place, and these, in turn, may give rise to infection of the lung. Pulmonary tuberculosis develops with remarkable frequency after antecedent *serous pleuritis*, whose tuberculous nature can be readily demonstrated by inoculation of the pleural exudate into the abdominal cavity of guinea-pigs.

Chronic pulmonary tuberculosis may develop at any *age*, and in either *sex*. The disease is rare on high *mountains*, but it is not correct to consider elevations of above 500 meters immune. The convergence of the population toward large cities, vitiation of the air by extensive factories, long hours of work in closed rooms, often with insufficient nourishment, neglect of prophylactic measures against infection—all of these are factors that favor the spread of the disease in modern life, and have made it a serious pestilence of the present time.

Anatomic Alterations.—Chronic pulmonary tuberculosis has a marked tendency to begin in the *apices of the lungs*. Only in children are the first symptoms believed to begin especially in the basal portions. The frequent commencement in the apices of the lungs depends, in my opinion, upon the fact that these portions of the lungs take little part in respiration, so that tubercle-bacilli readily become lodged there, and their growth is favored by the deficient renewal of air. The scanty provision of the apices of the lungs with blood must also be considered as a factor favorable to infection. Some anatomists, it is true, maintain that the apices of the lungs participate with especial activity in respiration, and that thereby inspired tubercle-bacilli are forced into those situations. Often the apex of one lung is first attacked. In the further course of the disease the chronic tuberculous lesions extend progressively and involve the entire upper lobe, then also the remaining portions of the lung; but the upper lobes are always likely to exhibit the most profound alterations. The extension of the disease is favored in part by the inspiration during the act of coughing of sputum containing tubercle-bacilli into healthy bronchioles and alveoli.

The distinctive alterations in the lungs consist in the formation of yellowish, cheesy masses, resulting from round-cell proliferation by a process of desiccation and necrosis. Tubercle-bacilli are frequently wanting in the necrotic masses, and often are present only in recently diseased areas. Nevertheless, the cheesy masses also must contain infective matters, probably the as yet undemonstrable spores of tubercle-bacilli, as their infectivity can readily be demonstrated by experiments on animals. At the beginning the tuberculous masses almost represent a jelly-like, translucent structure, so that the designation *gelatinous pneumonia* has been employed. The dry, cheesy tissue has a tendency to undergo *softening* and *breaking down*. The softened material is in part absorbed, but is largely expectorated with the sputum, and in this way the formation of cavities results—**tuberculous caverns.** Not rarely one lung contains numerous cavities that transform the viscus into a multilocular structure, while in other instances but a few disseminated caverns are encountered. Also, a single cavity has sometimes formed, but which, under some circumstances, may occupy almost the entire lobe of a lung. At the same time the cavity will not rarely have advanced to just beneath the pulmonary pleura. The latter will then generally be thickened in consequence of chronic inflammation and be firmly adherent to the costal pleura. The lining membrane of the cavern is at first ragged and shreddy, but subsequently it becomes smooth. Often the cavity is traversed by strands that are nothing more than blood-vessels, which have for a longer time offered resistance to the destructive process. The lining membrane of caverns often exhibits nodular dilatations—*aneurysms* of blood-vessels—which in case of rupture give rise to profuse hemorrhage.

On *microscopic examination of the lungs* it will be found that the inflammatory and cheesy masses are present both in the pulmonary alveoli and between them, so that generally intra-alveolar and interalveolar alterations are situated side by side. In the past it has often been a matter of discussion whether the first changes took place within the pulmonary alveoli or in the inter-alveolar connective tissue; obviously both conditions occur. In cases of inhalation-tuberculosis the alterations will probably first be intra-alveolar, while in cases of secondary—hematogenous—pulmonary tuberculosis they will probably begin in the inter-alveolar connective tissue. The round-cells that constitute the tuberculous tissue are derived in part from the colorless corpuscles of the blood, but another portion is derived from proliferation of the tissue-cells. Grayish, translucent tubercles are not rarely encountered in the vicinity of caseous masses. The **blood-vessels** within the area presenting tuberculous changes frequently exhibit endarteritic thickening. Tubercles also are frequently present in their walls. The **bronchi** likewise are involved in the disease. The peribronchial connective tissue is increased in amount, tuberculous-caseous masses form in it, and also upon the bronchial mucous membrane tubercles frequently develop, the breaking down of which gives rise to tuberculous ulceration of the mucous membrane. Often the bronchi are dilated and filled with cheesy masses, and care must be taken not to mistake bronchial dilatations for true pulmonary cavities.

Tuberculous-caseous foci, if not too large in extent, are occasionally enclosed within a capsule of black or slate-colored connective tissue. The cheesy mass gradually diminishes in size, and not rarely undergoes calcification. Some foci disappear wholly, and are replaced by so-called **slaty cicatrices,** which owe their origin to chronic interstitial pneumonitis, and persist throughout life.

Among the remaining visceral alterations attending chronic pulmonary tuberculosis, **tuberculous and cheesy changes in other organs** are to be taken into consideration, often originating by the conveyance of tubercle-bacilli from the lungs. In addition, signs of profound *emaciation* are generally present. The heart-muscle particularly is remarkable for its smallness, flabbiness, and brownish discoloration—brown or pigmentary atrophy of the heart.

Symptoms and Diagnosis.—In the recognition of chronic pulmonary tuberculosis the **demonstration of tubercle-bacilli in the sputum** is the most reliable sign, although it should be clearly borne in mind that tubercle-bacilli can be expected only if tuberculous tissue has undergone softening and breaking down, and is admixed with the sputum. Also, in doubtful cases, the examination of the sputum must be frequently repeated, as, possibly, tuberculous masses may at times accidentally not be contained in the sputum. It is extremely rare for tubercle-bacilli constantly not to be found in cases of clinically well-marked pulmonary tuberculosis in spite of repeated and careful examination of the sputum, and this circumstance is to be explained only upon the assumption that softening and expulsion of cheesy masses do not take place.

To demonstrate the presence of *tubercle-bacilli* the sputum should be spread upon a white plate, and with forceps such portions are to be selected as are opaque and friable. One of these is placed upon a clean cover-glass, upon which a second cover-glass is superimposed, and the particle of sputum is compressed between the two into a uniformly thin layer. Then the cover-slips are slowly separated and are exposed to the air until the sputum has dried. Now the cover-slip is grasped between the thumb and the index-finger, and, with its sputum-surface upward, is slowly passed from five to ten times through an alcohol-flame or a gas-flame. Next, fresh *staining solutions* are prepared. Personally, I prefer slow staining for twenty-four hours to rapid staining with application of heat. In order to intensify the staining quualities of the aniline dyes aniline-oil or carbolic acid is added to the solutions of these dyes. I prefer the former, and fill the lower portion of a test-tube with aniline-oil, then add water until the tube is half full, agitate the mixture with the thumb applied to the mouth of the tube, and pass the solution through a paper-filter into a watch-glass. Then to this watery aniline solution an alcoholic solution of fuchsin is added so long as the solution remains clear. The previously prepared cover-glasses are now permitted to float upon the surface of this solution for twenty-four hours, with the sputum-surface downward. After the lapse of the period named the cover-glasses are transferred to a watch-glass containing absolute alcohol, to which from three to five drops of pure nitric acid are added. In this mixture all of the elements of the sputum, with the exception of the tubercle-bacilli, yield up the fuchsin. It is one of the tinctorial peculiarities of the bacilli that, although they take up the aniline stains with difficulty, they also retain them tenaciously when once they are stained. The preparation is given a more beautiful and distinct appearance if it be rinsed in water and be exposed for one or two minutes to the action of a solution of malachite-green or methylene-blue. After the cover-glasses have been rinsed in water, dried by being passed through a flame, and mounted in xylol-Canada balsam, the tubercle-bacilli will, under such circumstances, appear stained bright red, while all of the cells and such other bacteria as may be present will be stained green or blue, respectively (Fig. 77). A trained eye may be able to recognize tubercle-bacilli with a magnification of from 300 to 500. The bacilli appear as thin, delicate rods, which occasionally can be detected only after prolonged search, while in other instances they are almost uniformly distributed in innumerable amount over the entire field, or in places are collected together in groups. Not rarely they exhibit small, unstained areas (Fig. 77), that have been considered as spores.

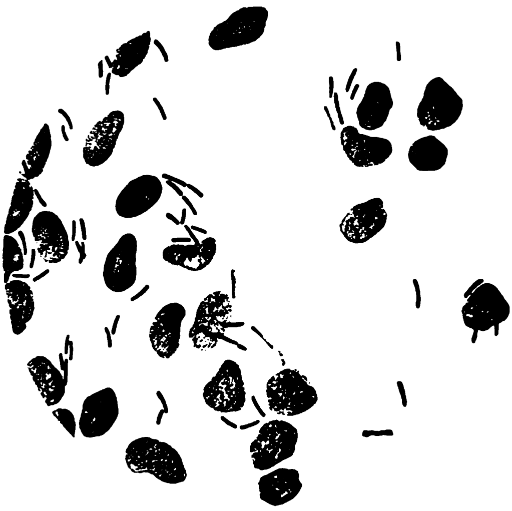


FIG. 77.—Tubercle-bacilli from the sputum in a case of pulmonary tuberculosis; fuchsin-malachite preparation; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

In addition to tubercle-bacilli, **elastic fibers** also should be looked for in the examination of the sputum. These also are most likely to be encountered in the opaque friable masses that have been mentioned as containing the tubercle-bacilli. They will be brought into view with especial distinctness if potassium [illegible] be added to a microscopic sputum-preparation, as a result [illegible] elements of the sputum are dissolved, while such elastic fibers as may be present stand out with especial distinctness. At times only isolated, or but a few, fibers are present, while at other times they exhibit a distinct alveolar arrangement (Fig. 78). The greater the number of elastic fibers found in the sputum the more active, naturally, will the destructive process in the lungs have been. In order to obtain elastic fibers from the sputum in the most perfect condition the expectorated matter, together with an equal amount of water and potassium hydroxid, should be heated to boiling in a beaker, and precipitation be permitted to take place. The sputum will thereby be converted into a fluid of watery consistence, in which everything but the elastic fibers are dissolved. The latter are deposited upon the bottom of the vessel as a sediment. It is advisable, further, for the purpose of more exact study, to decant the clear fluid carefully, and to centrifugate the sediment before it is submitted to microscopic examination.

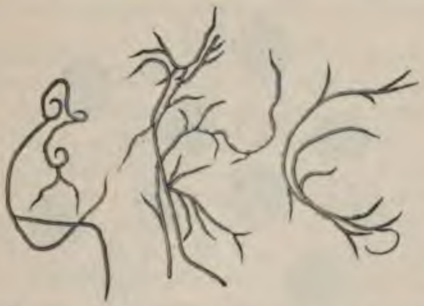


FIG. 78.—Elastic fibers from the expectoration in a case of pulmonary tuberculosis; magnified 275 times (personal observation).

Among the features of chronic pulmonary tuberculosis the sputum has been given first place, not only because the presence of tubercle-bacilli and elastic fibers constitutes a most certain sign of the disease, but because both of these structures occasionally appear almost as the first symptom of the disorder at a time when only doubtful, if any, physical changes in the lungs are demonstrable.

The **macroscopic appearances of the sputum** are under some circumstances likewise of diagnostic importance. At the beginning of the disease the expectorated matters occasionally possess a gelatinous consistency in correspondence with the appearance of the diseased portion of the lung, and on microscopic examination exhibit an exceedingly large number of alveolar epithelial cells, which are in a state of advanced fatty degeneration, and in many instances are converted into complete fatty granular cells. These cells readily undergo degeneration, and give rise to the formation of myelin-bodies (Fig. 79). When cavities have formed the sputum frequently acquires a coin-like appearance—*nummular sputum;* or a globular arrangement—*globose sputum.* Should globules of pus exhibit a tendency to sink to the bottom, the designation *sputa globosa fundum appetentia* has been employed. Naturally the diagnosis is not rarely attended with serious difficulty if no sputum is expectorated.

Chronic pulmonary tuberculosis begins insidiously, as a rule, and often the disease is not recognized in its early stages. Women are frequently treated for obstinate chlorosis in whom the anemia is only a result of concealed pulmonary tuberculosis. Chronic laryngeal catarrh also, as well as gastro-intestinal catarrh, is frequently dependent upon pulmonary tuberculosis. Some patients complain of rheumatoid muscular and articular pain, to which incipient pulmonary tuberculosis occasionally gives rise. Also, progressive emaciation without demonstrable cause often finds its explanation in insidious pulmonary tuberculosis. The conditions are especially suspicious if evening-fever is frequently present, and profuse sweating occurs at night. All of the manifestations mentioned should receive especial consideration if they occur in persons in whose family a number of cases of pulmonary tuberculosis have occurred, or who have associated a good deal with individuals suffering from pulmonary tuberculosis, or who exhibit the phthisical habitus.

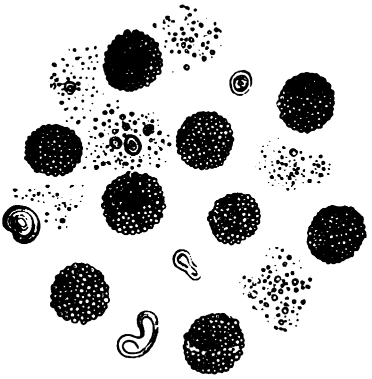


FIG. 79.—Alveolar epithelial cells involved in fatty degeneration: some filled with pulmonary pigment, others disintegrated; myelin-bodies also are present; magnified 275 times (personal observation).

Persons presenting the *phthisical habitus* are generally tall, with long, slender bones, flabby and imperfectly developed muscles, and a slight layer of subcutaneous fat. Generally, they are pale, although they exhibit an irritable vasomotor nervous system, so that they rarely become flushed on slight physical and mental exertion. The hair is markedly dry and scanty, the eyes peculiarly lustrous, almost staring, and the sclera glistening white.

Frequently the eyeballs are retracted and the eyelids surrounded by a bluish or reddish border. The teeth generally are noteworthy for their length, their bluish-white color, and a certain translucency. The neck is characterized by its length and thinness, so that it has been designated also swan's neck. The chest presents unusual length and width, but slight depth. The intercostal spaces are of unusual width. The junction between the manubrium and the body of the sternum presents an angular projection forward —the so-called angle of Ludwig; and, posteriorly, the separation of the median border of the scapulæ from the dorsal aspect of the chest is conspicuous. Such shoulder-blades have been compared with angel's wings, and they have been designated also alar or winged scapulæ. As the widening of intercostal spaces is attributed to weakness of the intercostal muscles, and the separation of the shoulder-blades to weakness of the serratus muscles, the phthisical chest has been designated also the *paralytic thorax.* The chest takes but little part in the respiratory movements, and even after deep inspiration, in comparison with a healthy chest, the impression is made as if it were in the stage of expiration. From this circumstance is derived the name *permanent expiratory thorax.* If pressure be made upon the anterior aspect of the chest, considerable resistance will generally be encountered, because the costal cartilages often become ossified quite early.

Physical examination of the lungs will disclose variable conditions in accordance with the pulmonary alterations present. In many cases it is significant to discover **local retraction of the chest,** which generally involves the supraclavicular fossa and the upper portions of the infraclavicular region. This is indicative of contraction of the underlying portions of the lung, and this in turn is frequently dependent upon tuberculous alterations in the upper portions of the lung. As a rule, the retracted portions of the chest exhibit also **slighter participation and a backwardness in the respiratory movements.** Under certain conditions, it is of importance to demonstrate that the **apex** of one lung extends to a **higher level** than that of the other. This can be done, naturally, only by percussion, although it should be borne in mind that occasionally also in healthy individuals a difference in the height of the apices of the lungs occurs. **Differences in the percussion-note,** particularly over the upper portions of the lung, are extremely valuable in diagnosis. At the commencement of the disease they are often quite slight, and their detection requires an experienced ear. It has always proved valuable to me to continue percussion without interruption during deep inspiration and expiration on the part of the patient, and to observe differences in the percussion-note between corresponding points upon the two sides of the chest. I attach considerable weight also to the sense of increased resistance yielded on percussion. If cavities have formed, the percussion-note is generally dull and tympanitic, occasionally, also, metallic, only at times being completely dull, when the cavity is filled with fluid. Wintrich's and the respiratory change in note also are not rarely audible, while interrupted Wintrich's and Gerhardt's change in note are less commonly encountered, because these require the presence of readily movable secretion in the pulmonary cavity.

For the recognition of incipient pulmonary tuberculosis certain **auscultatory phenomena** also are of importance. Unilateral **jerky breathing** over the apices, **rough vesicular respiration** in the same situations, and **prolonged expiration** are dependent upon catarrhal conditions in the smaller bronchi at the apices of the lungs, and these in turn are frequently due to beginning pulmonary tuberculosis. If the tuberculous process is further advanced, tuberculous infiltration and airlessness of the pulmonary tissue will be disclosed by bronchial breathing, in addition to dulness and increased vocal fremitus, and over smooth-walled cavities of at least five centimeters in diameter metallic or amphoric bronchial breathing will be audible. Almost always râles, generally moist or bubbling, are present, and from the airless tissue these receive a peculiar intensification that is designated ringing or consonating. Bubbling in large, smooth-walled cavities yields a metallic note.

In some cases pulmonary tuberculosis is wholly unattended with fever; other patients exhibit fever only at times. In still others, however, persistent **fever** is present, which is characterized by marked exacerbations at night, whereas the bodily temperature may be normal or subnormal in the morning. Such fever, attended with steep temperature-curves, has been designated *hectic fever*. It resembles that which attends suppuration, and is only a suppurative fever, induced by the extrance into the blood of pyogenic cocci from the purulent and softened cheesy masses. The fever often is initiated will chilliness, which occurs most frequently about midday. The cheeks become vividly red in the malar region, and this has been designated *hectic flush*.

Many patients suffer from distressing **night-sweats**, which occur especially during the first hours after midnight, and not only disturb sleep, but also cause great weakness. In consequence in part of the sweating molds thrive readily upon the skin. For this reason brownish spots appear upon the skin in many cases of pulmonary tuberculosis, particularly upon covered portions of the body (chest, abdomen, back), which can be readily removed with the finger-nail. If the scales be cleared up with potassium hydroxid, fungous spores and filaments of the Microsporon furfur will come into view (p. 221, Fig. 44). In short, pityriasis versicolor is frequently encountered in cases of pulmonary tuberculosis.

Gastric disturbances are extremely common in cases of pulmonary tuberculosis. Insuperable anorexia appears, and if in spite of it the ingestion of food is insisted upon vomiting will take place. These manifestations are dependent in part upon weakness of the muscular coat of the stomach, and in part upon deficiency or absence of hydrochloric acid from the gastric juice.

The *course of pulmonary tuberculosis* is generally chronic. An acute course with persistent fever is less common, and among the laity is known also as *galloping consumption* or *florid pulmonary*

[illegible]

[illegible] the

patients are seized with extreme dyspnea with the onset of pneumothorax, but otherwise the condition develops insidiously and without noteworthy symptoms, and, therefore, may readily be overlooked, particularly if the pneumothorax be sacculated. Suspicion should always be aroused if the patient persistently lies upon one and the same side.

Secondary tuberculous complications may occur in any organ. At times infection takes place immediately through the sputum containing tubercle-bacilli. At other times tubercle-bacilli are conveyed from the lungs through the blood-vessels and the lymphatics. In the latter event, therefore, the complications are, in a certain sense, metastatic—embolic. Tuberculous involvement of the *larynx*, the *pharynx*, the *buccal mucous membrane*, and the *tongue* is probably attributable, in the majority of cases, to the fact that portions of the sputum adhere, in the act of expectoration, to the structures named, and thereby give rise to infection. This, however, does not exclude the possibility that occasionally metastatic infection also occurs. Contamination of cutaneous wounds with the expectoration may give rise to tuberculous lesions in the skin; picking of the nose with contaminated fingers to lesions in the nares, etc. The swallowing of the sputa may readily give rise to infection of the *lymphatic follicles of the intestinal mucous membrane*, upon which tuberculous ulcers may develop. The patients then complain frequently, but not constantly, of pain in the abdomen, and they suffer from irregularity in the action of the bowels, either from constipation or more frequently from diarrhea. Tubercle-bacilli not rarely can be demonstrated in the stools, although it should be borne in mind that these may be derived also from swallowed sputum in the absence of intestinal tuberculosis.

Embolic secondary tuberculosis, in the course of chronic pulmonary tuberculosis, may occur, in addition to the organs already named, also in any other viscus; thus, in the meninges, the urogenital organs, the peritoneum, etc. By no means rarely death occurs in cases of pulmonary tuberculosis from tuberculous meningitis or general miliary tuberculosis.

Among the *complications resulting from excessive marasmus* are *cachectic edema* and *marantic venous thrombosis*, the latter usually involving the distribution of one of the femoral veins, most frequently that of the left femoral vein. In some cases *purpura* occurs, occasionally covering the skin of the leg profusely, particularly if the patient has been upon his feet for a long time. As after all chronic suppurations, so also after chronic tuberculous suppuration of the lungs, *amyloid degeneration* may result. Under such circumstances amyloid disease of the kidneys will be indicated by marked edema of the lower extremities and the presence of a considerable amount of albumin in the urine. Amyloid degeneration of the intestinal mucous membrane is attended with severe diarrhea that is difficult to control. Amyloid spleen and amyloid liver can be recognized from the enlargement and the marked induration of the diseased organs. Occasionally chronic *parenchymatous nephritis* develops in the sequence of pulmonary tuberculosis, and this is not rarely associated with amyloid disease of the kidneys.

Prognosis.—There is not the slightest doubt that chronic pulmonary tuberculosis is susceptible of cure. In the first place, clinical experience is in favor of this view, and, besides, encapsulated, calcified, or completely absorbed tuberculous foci are encountered by no means rarely at the apices of the lungs in the cadaver. Nevertheless, the prognosis of pulmonary tuberculosis is grave, as the majority of patients are not capable of surviving until cure is effected, and many come under the observation of the physician only at too late a period, and a cure can no longer be hoped for in view of the extent of the changes in the lungs. The prognosis is aggravated by all *complications*. Naturally, some of these are of more grave significance than others.

Treatment.—There is as yet no *specific remedy* for the treatment of chronic pulmonary tuberculosis. All of the agents recommended as such have soon proved useless. The most trustworthy remedies in the treatment of pulmonary tuberculosis **are pure air and nutritious diet.** The pure air of high altitudes is especially beneficial for the patient, and without doubt the results of treatment at the high-altitude resorts of Switzerland, particularly Arosa, Davos, and Leysin, are the best. Residence at these places is especially to be recommended during the winter. In summer, for residence during which Switzerland contains likewise many high-altitude resorts (Beatenberg, Churwalden, Dissentis, Klosters, Tennigerbad, etc.), Germany also offers resorts in the Schwarzwald, in the Bavarian mountains (Kreuth, Reichenhall), and in the Hartz mountains. Patients who are inclined to excesses should seek admittance into *sanatoria*, thus in private institutes, such as exist not only in Arosa and Davos, but also in Germany (Falkenstein, Görbersdorf, Honnef), where they will be constantly under medical observation. The earlier climatic treatment is instituted, and the longer it is persisted in, the more permanent and the better will be the results. Not much can be accomplished in the treatment of pulmonary tuberculosis during the customary four weeks of bathing. Most patients must continue the treatment throughout the winter, and repeat it in subsequent years; or, still better, remain uninterruptedly for years in health-resorts.

Persons with laryngeal tuberculosis and a tendency to hemoptysis are not suitable for high-altitude treatment, and will do better to seek the *southern health-resorts*. Among these Madeira, Teneriffe, and Ajaccio in Corsica, are especially to be recommended. Next the health-resorts upon the Italian and French Riviera, especially Nervi, Rapallo, St. Remo, and Mentone should be mentioned.

As climatic health-resorts are accessible only to well-to-do patients, a beginning has been made in recent years in the erection of *public sanatoria* in which poor patients can be treated gratuitously, or for a small amount. The money necessary for the erection of such structures has been derived principally from private sources, and in isolated instances also from beneficial organizations.

In all climatic treatment it is especially important for the patient to live as much as possible in the fresh air, and this, naturally, will be possible only if the resorts are free from fog and the wind is quiet. Protected halls in which the patient can lie recumbent throughout the day have been provided at such sanatoria.

As the sea-air is extremely pure, long *sea-voyages* have been recommended to patients with means, and, particularly in America, sailing-vessels have been equipped for the reception of tuberculous patients. The anticipated results from long-continued *residence at the seaside* have not been realized.

In every case of pulmonary tuberculosis an abundance of *food* should be given, rather more than is actually needed. The diet may be a mixed one. One or two liters of *milk* especially are to be recommended daily, given in small amounts at intervals, and to be used only after previous thorough boiling. If milk be badly borne, or is not readily taken, a teaspoonful of cognac may be added to every glass or cup. If the aversion to milk be insuperable, efforts to conceal its taste should be made with other articles of food, and it may be administered in the form of milk-cocoa, milk-coffee, milk-tea, rice-gruel, or oatmeal-gruel. The use of *kefyr* also may be recommended. The employment of *fat* in the form of butter, fat meat, fat ham, and fat sausage may be advised. Especially in the past *codliver-oil*, 25.0 (6 drams) morning and evening, was much prescribed, but it should be administered only if the patient otherwise takes too little fat; it should also be given only in the winter, because in the summer it readily causes loss of appetite. From time to time, about every four weeks, intermissions in the administration of codliver-oil should be observed. It is noteworthy in cases of pulmonary tuberculosis, that while wine and beer may be permitted, care should be taken to avoid excess. Half a liter of wine and half a liter of beer daily are sufficient.

Resort to drugs freely should be had only in case of necessity, if special symptoms seem to demand their use. If the patients are anemic, *preparations of iron* should be employed, as, for instance:

℞ Iron lactate,
Sodium bromid, each, 10.0 (2½ drams);
Arsenous acid, 0.1 (1½ grains);
Powdered althea-root, sufficient to make 100 pills.—M.
Dose: 2 pills thrice daily after meals.

Excessive accumulation of secretion in the air-passages is not rarely an indication for the employment of *expectorants*. In order to diminish the secretion *balsamics* and *disinfectants* have largely been used, particularly creosote and preparations of guaiacol; but the belief that these remedies are capable of exerting a specific influence upon pulmonary tuberculosis is, in my opinion, based upon a gross error. Personally I like to use benzosol, which is generally better borne by the patient than creosote or guaiacol:

℞ Benzosol, 0.5 (7½ grains);
Eleosaccharum of peppermint, 0.3 (4½ ").—M.
Make 10 such powders.
Dose: 1 powder thrice daily after meals.

When irritative cough is severe *narcotics* may be administered, as, for instance:

℞ Morphin hydrochlorate, 0.1 (1½ grains);
Cherry-laurel water, 10.0 (2½ fluidrams).—M.
Dose: From 5 to 10 drops when the cough is troublesome.

For the relief of profuse night-sweats friction of the skin every evening with a coarse towel that has been dipped in a vessel of water to which a few tablespoonfuls of vinegar have been added may be recommended. The towel is first thoroughly wrung out until it no longer drips. The rubbing should be continued for only a few seconds (from fifteen to thirty), and then be followed by friction with a dry towel. In addition, *atropin* may be prescribed:

℞ Atropin sulphate, 0.005 ($\frac{1}{15}$ grain);
Extract of licorice, sufficient to make 10 pills.—M.
Dose: 1 or 2 pills to be taken at night.

In the *prophylaxis of chronic pulmonary tuberculosis* it is especially important to give thorough consideration to *disinfection of the sputum*. Patients suffering from pulmonary tuberculosis should never expectorate upon the floor or into a handkerchief, but into vessels or into small, readily portable flasks that can be closed and are filled with a 5 per cent. solution of carbolic acid. In order to avoid the evil effects to which the matters expelled by the coughing may give rise, patients with pulmonary tuberculosis have been advised to wear *mouth-masks*. The *clothing* and the *linen* of tuberculous patients should be thoroughly sterilized in live steam before they are used by others. Rooms that have for a long time been occupied by patients suffering from pulmonary tuberculosis should first be disinfected before they are occupied by healthy persons.

Persons with the *phthisical habitus* should from youth be hardened in an intelligent manner by cold sponging and friction, and be strengthened by appropriate gymnastic exercises. *Marriage* is not without danger in cases of pulmonary tuberculosis. In women tuberculous disease of the lungs frequently undergoes rapid exacerbation during pregnancy, and particularly after the puerperium; and in men, likewise, the excitements of married life are a serious disadvantage. The prophylaxis includes also the suppression of *tuberculosis in cattle*, as this is often the source for tuberculosis in human beings.

CHRONIC LARYNGEAL TUBERCULOSIS.

Etiology.—Chronic laryngeal tuberculosis, also designated *consumption* or *phthisis of the larynx*, is much less common than pulmonary tuberculosis. While the latter is most frequently of primary origin, chronic laryngeal tuberculosis is almost always a **secondary** disease, which generally complicates antecedent pulmonary tuberculosis, less commonly chronic tuberculosis of other organs. The infection of the larynx occurs, as a rule, through the lodgment in the act of expectoration of sputum containing tubercle-bacilli upon the laryngeal mucous membrane, although in other instances infection may take place also through the intermediation of the blood-vessels and the lymphatics. Long-continued and loud speaking, inhalation of dust and irritating vapors, tobacco-smoking, and antecedent syphilis are properly considered *contributory causes* favoring infection. In the last-named event *mixed infection with syphilis and tuberculosis* occasionally occurs. From the nature of the contributory causes it will be understood that chronic laryngeal tuberculosis is about four times as common in men as in women. Primary laryngeal tuberculosis is dependent upon the same causative factors as primary pulmonary tuberculosis.

Symptoms and Anatomic Alterations.—Athough laryngeal tuberculosis often develops insidiously at first, the symptoms soon become so marked that the disorder justly is greatly feared. Most frequently **disturbances in phonation** appear, varying between an obscure and hoarse voice and complete aphonia. The act of speaking often not only is attended with strain, but also induces pain in the larynx. Quite frequently **pain in swallowing** is present, if the arytenoid cartilages and the epiglottis are involved, and are mechanically irritated by each act of swallowing and each bolus of food. Should the tuberculous alterations extend deeply, *laryngeal perichondritis* and its sequels may occur. The expectoration not rarely becomes quite abundant, and acquires an offensive odor. The patients are harassed day and night by severe cough. *Pressure upon the larynx* and displacement thereof often excite diffuse or circumscribed pain. The severe pain induced by the ingestion of food causes many patients to refrain from eating, and under such circumstances death results from exhaustion within a short time. In some cases death results from *acute edema of the glottis* or from *suffocation*, if rupture of perichondritic abscesses takes place.

The *anatomic alterations* can best be studied macroscopically during life with the aid of the laryngoscope. If the **epiglottis** is the seat of tuberculous alterations, it is not rarely converted into a roundish and cylindrical body as thick as a finger, generally with an irregular and nodular surface. Should caseation and breaking down of the tuberculous infiltrate take place, the epiglottis occasionally becomes eroded down to its pedicle, so that only a slight remnant persists. Occasionally tuberculous alterations have taken place especially upon the **false vocal bands**, where I have observed them to occur in follicles, and give rise to the formation of deep, crater-like ulcers. Often the **arytenoid cartilages** and the **posterior attachments of the vocal bands** are the seat of tuberculous alterations and ulceration, and also the **interarytenoid space** constitutes a favorite seat for tuberculous disease of the larynx. Occasionally tuberculous ulceration extends from the larynx to the tracheal or the bronchial mucous membrane, less commonly upward to the pharyngeal mucous membrane. **Tuberculous polypoid proliferations** appear in the larynx, at times at the margins of the ulcers.

Microscopic examination discloses infiltration of the diseased areas with round-cells, and here and there giant-cells and tubercle-bacilli.

Diagnosis.—The recognition of laryngeal tuberculosis is not difficult, and can be made with certainty if *tubercle-bacilli* are demonstrable in the discharge from the ulcers obtained from the larynx with a suitable brush. Naturally, one must assure himself that the tubercle-bacilli are not possibly derived from remnants of sputum. The diagnosis whether laryngeal tuberculosis is primary or secondary is occasionally attended with great difficulty, because latent pulmonary tuberculosis may be associated with extensive laryngeal tuberculosis.

Prognosis.—Recovery from laryngeal tuberculosis takes place but seldom, so that the prognosis is unfavorable in any connection.

Treatment.—Persons with tuberculosis in other organs should guard against injurious influences capable of affecting the larynx, particularly loud and long-continued speaking, smoking, and inhalation of dust and irritating gases. If the tuberculous lesion is not too extensive, attempts may be made to destroy all of the tuberculous tissue by deep **cauterization.** For this purpose concentrated lactic acid especially has been recommended. If the alterations are extensive, resort may be had to **scraping of the larynx—curettement.** I have repeatedly observed marked improvement, particularly amelioration in the subjective symptoms, after **tracheotomy**. In order to mitigate the severity of the pain, and especially to render swallowing possible, **narcotics** must be administered, among which application with a brush of a solution of cocain to the entrance of the larynx may be recommended:

℞ Cocain hydrochlorate,	1.0 (15 grains);
Alcohol,	2.0 (30 minims);
Distilled water,	8.0 (2 fluidrams).—M.

To be applied with a brush thrice daily before meals.

If food frequently enters the larynx in the act of swallowing, **nourishment should be given by means of the esophageal tube.**

CHRONIC PHARYNGEAL TUBERCULOSIS.

The **etiology** of chronic pharyngeal tuberculosis is identical with that of laryngeal tuberculosis. The condition, therefore, is generally one of **secondary tuberculosis**, occurring most frequently in cases of pulmonary tuberculosis, and being due to inoculation of the pharyngeal mucous membrane with expectorated sputum containing tubercle-bacilli. It has already been mentioned in the preceding section that not rarely *chronic laryngeal and pharyngeal tuberculosis are present simultaneously*. The disorder occasionally develops quite insidiously, and it may possibly be discovered accidentally on inspection of the pharyngeal structures. In other instances, however, the patients complain of severe *pain in the pharynx*, which is increased especially on swallowing and in speaking. Not rarely the pain radiates toward the region of the ear.

Generally the **anatomic alterations** consist in sharply circumscribed **ulcers**, which are situated with especial frequency on the summit of the anterior palatine arch, and upon one or both sides, and by coalescence they give rise to a number of excavations. The tuberculous nature of these ulcers will be made certain by the detection of *tubercle-bacilli* in the discharges. I have also observed **miliary tubercles** in the margins of the ulcer in a number of instances. The ulcers may undergo healing, but this occurs but rarely.

Treatment.—Attempts should be made by deep **cauterization** with concentrated lactic or carbolic acid to destroy all tuberculous tissue, and then to induce firm cicatrization. **Scraping of the ulcers** also may be useful.

CHRONIC INTESTINAL TUBERCULOSIS.

Etiology.—Chronic intestinal tuberculosis is generally of *alimentary* origin or due to the eating of tuberculous *food*. It occurs with especial frequency as **secondary tuberculosis** in cases of pulmonary tuberculosis in which the patients are in part in the habit of swallowing the sputum. It may occur as *primary intestinal tuberculosis* in consequence of the ingestion of milk and milk-products (butter, cheese) derived from tuberculous animals. It is, therefore, encountered most frequently in countries in which tuberculosis is prevalent among cattle. It occurs, also, with unusual frequency in children nourished with cows' milk.

Symptoms and Diagnosis.—Intestinal tuberculosis, even if extensively developed, may be unattended with abnormal manifestations—*latent intestinal tuberculosis*—and may be discovered accidentally on post-mortem examination. Occasionally it is attended with severe **abdominal pain—enteralgia**—which is often paroxysmal and colicky in nature, at times occurring with a certain regularity during the first hours following midnight. The pains are generally referred to the region of the right iliac fossa, because the tuberculous alterations are principally situated in the lowermost portions of the ileum. Also, pressure in this region generally gives rise to severe pain. There is often **irregularity in the action of the bowels,** occasionally obstinate constipation, often, however, severe, uncontrollable diarrhea. The demonstration of tubercle-bacilli in the stools is especially valuable in establishing the diagnosis; but this is of unequivocal significance only if assurance exists that the patients have not swallowed the sputum. Occasionally **intestinal hemorrhage** and **perforation of the bowel,** and their sequelæ, occur unexpectedly if a blood-vessel is eroded or the intestinal wall is perforated in the destructive process attending the tuberculous alterations. There is, also, danger of *diffuse* or *circumscribed peritonitis* if the ulceration has too closely approached the serous coat of the bowel. Tuberculous ulceration in the vermiform appendix or the cecum occasionally gives rise to *perityphlitis* and *paratyphlitis*. *Stricture of the bowel* and *intestinal obstruction* occasionally occur as *sequelæ*. While the *diagnosis* of chronic intestinal tuberculosis is in and of itself attended with serious difficulty, it is, besides, not always easy to differentiate primary from secondary intestinal tuberculosis; for, in the presence of simultaneous disease of the intestine and the lungs, the conditions may be such that the lungs are infected by tubercle-bacilli conveyed from the intestine.

Prognosis.—The prognosis of chronic intestinal tuberculosis is serious, as under all circumstances the disease favors progressive exhaustion, apart from such serious complications as may arise. Nevertheless, healing of tuberculous intestinal ulcers is possible, and I have observed this condition repeatedly on post-mortem examination.

Anatomic Alterations.—Intestinal tuberculosis develops in the **lymphatic follicular apparatus** and the **lymphatic vessels of the intestinal mucous membrane.** The localization of the anatomic alterations corresponds, therefore, with that in cases of typhoid fever. They are thus most marked, and often exclusively present in the lower portions of the ileum. They are much less commonly present in the colon. Both the solitary follicles and Peyer's patches are involved in the tuberculous process, the latter at first only in certain portions. Often the areas that are the seat of tuberculosis can be recognized from without, as the affected portion of intestine presents a blackish or reddish appearance, also is frequently retracted inward, and feels hard on palpation. Miliary tubercles also are frequently present beneath the intestinal serosa in the vicinity of the alterations described. If the intestine be opened, *tuberculous ulcers* in greater or lesser number and extent will generally be exposed, and which, in contradistinction from typhoid intestinal ulcers, are not arranged with their longitudinal axis parallel to that of the intestine, but frequently follow the lumen of the bowel in an annular manner, in accordance with the course of the intestinal lymphatics.

The intestinal ulcers resulting from destruction of the solitary follicles represent small, generally crater-like, deep losses of tissue upon the intestinal mucous membrane; while the ulcers that form upon Peyer's patches are more extensive, and often of irregularly excavated form. The ulcers also often pass beyond the actual limits of Peyer's patches, extending along the lymphatics to the adjacent intestinal mucous membrane. The margins of the ulcers are generally elevated like a wall, sharply defined, and here and there covered with miliary and cheesy tubercles. The base of the ulcer is generally formed by the muscular coat of the bowel, whose fibers can often be observed with great distinctness.

Not rarely it will be found that tuberculous ulcers of the intestine are cicatrized in some places while they are extending in others, although complete cicatrization and cure also take place, particularly in smaller ulcers. Generally these healed areas are conspicuous for their dark discoloration, resulting from transformed hemoglobin. Larger tuberculous ulcers that have healed, or are in process of healing, exhibit a marked tendency to cicatricial contraction, and *stricture of the bowel* occasionally results therefrom, diminishing the lumen of the intestine in greater or lesser degree in several places.

If the commencement of intestinal tuberculosis be more carefully investigated, it will be found that miliary tubercles first appeared in the enlarged lymph-follicles, gradually undergoing caseation and destruction. Adjacent ulcers coalesce and form progressively larger ulcers. Often the related *mesenteric lymph-glands* also are tuberculous, and they are not rarely converted into large caseous nodules. The retroperitoneal lymphatic glands also are at times transformed into cheesy packets, which occasionally can be felt during life through the abdominal wall as

Treatment.—For individuals with intestinal tuberculosis an easily digestible, principally liquid, **diet** should be prescribed. The use of vegetables and fruits especially requires care. In the presence of severe pain a hot **cataplasm** should be applied to the abdomen, and, if necessary, **subcutaneous injections of morphin** should be given:

℞ Morphin hydrochlorate, 0.3 (4½ grains);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: 8 minims for subcutaneous injection.

Should diarrhea exist, the injections of morphin will often exert a constipating influence by allaying the violent intestinal movements induced reflexly from the intestinal ulcers. **Regulation of the bowels** is one of the principal indications in the presence of intestinal tuberculosis. In accordance with existing conditions resort will at times be had to *laxatives*, at other times to *styptics*. Under the latter conditions bismuth salicylate especially may be recommended:

℞ Bismuth salicylate, 0.5 (7½ grains);
Powdered opium,
Saccharin, each, 0.02 (⅓ grain).—M.
Make 10 such powders.
Dose: 1 powder every three hours.

Resection of the intestine has been successfully undertaken for the relief of *stricture of the bowel*.

As a *prophylactic* measure individuals suffering from pulmonary tuberculosis should be admonished not to swallow the *sputum*. In addition, only thoroughly boiled *milk* should be used in order to render harmless any tubercle-bacilli that may be present, and efforts should be directed toward the eradication of *tuberculosis in cattle*.

CHRONIC TUBERCULOSIS OF THE KIDNEYS AND THE BLADDER.

Etiology.—Chronic tuberculosis of the urinary organs is generally **secondary**, and is most frequently a complication of primary tuberculosis of the sexual organs. The condition is designated also *urogenital tuberculosis*. In men chronic tuberculosis of the epididymis, the seminal vesicles, or the prostate gland, is with especial frequency an antecedent condition; while in women the oviducts and the ovaries are not rarely first involved. Naturally, secondary tuberculosis of the urinary organs may complicate tuberculosis of other viscera, as of the lungs, the bones, and the joints. *Primary tuberculosis of the urinary organs* is observed much less commonly. Under such circumstances infection through sexual intercourse with persons suffering from tuberculosis, particularly of the lungs, the urinary or the sexual organs, will have to be taken into consideration. In cases of pulmonary tuberculosis the seminal fluid not rarely contains tubercle-bacilli even when the urogenital organs are free from tuberculosis. The channels of infection cannot always be recognized with desirable certainty. Thus, I have observed tuberculosis of the urinary organs in a number of instances in children without being able to discover a source of infection.

Anatomic Alterations.—Tuberculosis of the urinary organs either begins in the kidney, and extends thence to the pelvis of the kidney, the ureter, and the bladder, or it originates in the bladder and gradually invades the kidneys. In the first event the designation *descending*, and in the latter, *ascending, tuberculosis* of the urinary organs, has been employed. Whether the one or the other variety is the more common cannot, at the present time, be decided with certainty. Further, it may also happen that tuberculosis is confined to the kidneys or the bladder.

Chronic tuberculosis of the kidneys generally begins in the papillæ of the organs, then extends more and more deeply into the medullary structure, and finally gives rise to caseation and purulent destruction of the renal cortex. Often there develop, in linear arrangement, at first grayish, subsequently yellowish tubercles, from the size of a lentil to that of a pea, which have in places become confluent. Should these undergo softening and destruction, the caseous tissue slowly breaks down, so that the papillæ of the kidney are wholly destroyed. On section, the kidney finally is transformed into an organ that contains in place of the papillæ deeply excavated spaces, generally filled with cheesy matter. If the renal cortex is actively involved in the tuberculous process, the diseased kidney may be increased in size and weight to more than ten times the normal, and in advanced cases it represents a fluctuating sac, formed by the capsule of the kidney filled with yellowish masses resembling brain-matter, and in which it is scarcely possible to demonstrate relatively well preserved renal tissue.

Not rarely extensive tuberculous alterations have taken place also upon the **mucous membrane of the pelvis of the kidney.** The mucous membrane is thickened in consequence of tuberculous proliferation, and often involved in caseation and destruction. Frequently the pelvis of the kidney is greatly dilated. The **ureters** not rarely appear as rigid tubes with thick walls, which in places exhibit nodular swellings. On incision their mucous membrane will be found thickened from the presence of tuberculous tissue, and here and there disintegration of the tuberculous masses has taken place, with the formation of tuberculous ulcers. Similar alterations are observed in the **bladder.** The tuberculous ulcers of the vesical mucous membrane are generally characterized by their sharp limitation. The margins appear thrown up like walls, and not rarely exhibit tubercles. The urine that has collected within the bladder is generally characterized by marked turbidity and a disagreeable odor.

Symptoms and Diagnosis.—As in the diagnosis of other varieties of tuberculosis, the *demonstration of tubercle-bacilli in the urinary sediment* is decisive also in the recognition of chronic tuberculosis of the urinary organs. Although with the aid of the centrifuge and the increased concentration of the urinary sediment therefrom possible, it has become considerably easier to detect tubercle-bacilli in the urinary sediment; nevertheless prolonged and continued search is not rarely necessary before the bacilli are found. It should be borne in mind that, as compared with the sputum, the tubercle-bacilli in the urine are distributed throughout much larger quantities of fluid. Especial consideration should be given to cheesy particles in the urinary sediment. Occasionally only single and isolated tubercle-bacilli are encountered, while in other instances they are present in plug-like, dense aggregations. Care should be taken, further, to avoid confounding tubercle-bacilli with smegma-bacilli, to which they bear a great morphologic and tinctorial resemblance.

If all attempts to detect tubercle-bacilli in the urine remain fruitless, inoculations of the urinary sediment into the abdominal cavity of guinea-pigs, or into the anterior chamber of the eye of rabbits, may be practised, and after the lapse of from four to six weeks observation be made to see whether the guinea-pigs have become tuberculous, or if tubercles have developed upon the iris in the rabbits.

Chronic tuberculosis of the urinary organs, especially of the kidney, occasionally begins unexpectedly with **bloody micturition —hematuria.** The clinical picture all the more resembles that of acute hemorrhagic nephritis because the urine not rarely contains tube-casts, and a larger amount of albumin than the amount of blood present would account for. The hematuria occasionally proves extremely obstinate. Nevertheless, tuberculosis of the kidney should be thought of in the presence of acute nephritis of undetermined origin. I have, under such circumstances, not rarely found tubercle-bacilli in the urinary sediment. In other cases the first symptoms appear under the clinical picture of *chronic purulent catarrh of the bladder.* The patients complain of severe vesical tenesmus, with pain in micturition, and turbid and generally acid urine is voided, which, on standing, precipitates a grayish-green sediment of pus-corpuscles, often several millimeters thick. Suspicion should be especially aroused in the case of children, in whom chronic catarrh of the bladder, not dependent upon tuberculosis, is rather an unusual condition. Further, the symptoms of catarrh of the bladder may occur also in association with uncomplicated renal tuberculosis, as the bladder is extremely sensitive to the action of urine of abnormal composition. Th presence in the urinary sediment of large numbers of desquamate pavement epithelium from the mucous membrane of the bladde would be in favor of involvement of the bladder. Occasionall the urine attracts attention on account of the odor of hydroge sulphid—rotten eggs—or of a putrid odor. Connective-tissue fiber also are occasionally present in the urinary sediment, and eve elastic tissue from the disintegrated structures.

In the *diagnosis of renal tuberculosis* attention should b directed to **local alterations in the kidneys,** particularly to spontaneous pain, and **pain on pressure.** Frequently renal tuberculosi begins upon one side, and accordingly pain will be present onl over one kidney. In advanced cases *increase in the size of th kidney* is not rarely demonstrable. It may also happen that th kidney varies in size, and that enlargement is coincident wit attacks of pain that wholly resemble *renal colic.* These conditions result from the detachment of cheesy masses of kidney o considerable size that from time to time occlude the ureter an give rise to *paroxysmal hydronephrosis.* During such attacks th urine becomes scanty and clear, because it is secreted only by th healthy kidney. Not rarely chilliness, even a chill, fever, an vomiting are observed at such times, symptoms of *urinary septicemia* resulting from absorption of urine containing pus.

Tuberculosis of the ureter can occasionally be recognized o examination through the rectum or the vagina, if the rigid urete frequently presenting nodular enlargements, can be felt upon th posterior wall of the bladder. The most reliable evidence wit regard to *tuberculous alterations in the bladder* will be afforded by th endoscope, which, it is true, is as yet only employed by specialists. Further, endoscopy is occasionally of importance also i the recognition of unilateral tuberculosis of the kidney, the endoscope being adjusted for observation of the ureters and it bein noted whether turbid urine escapes from one ureter, and whic Also catheterization of the ureters has been practised of late ; b this means it is possible to collect separately and examine bacteriologically the urine secreted by each kidney.

Patients with chronic tuberculosis of the urinary organs occasionally present a healthy and blooming appearance. If, howeve the disorder has existed for a considerable time, *emaciation* an *pallor* generally do not remain absent. The *bodily temperatur* often remains unaffected for a long time, although irregula febrile movement is not rarely observed. The *duration of th disease* is occasionally protracted over several years. Death results most frequently in consequence of progressive *exhaustion* an *urinary septicemia.*

Among the *complications,* the occurrence of *general milia tuberculosis* may be mentioned. [illegible] ally *rupture of the di* *eased organ* takes place. Rupture externally of caseous-tuberculous masses from the kidney will be followed by the development of *paranephritis* or *perinephritis*, or of a *fistula of the kidney* or of the *pelvis of the kidney*, while rupture into the peritoneal cavity generally gives rise to rapidly fatal *perforative peritonitis*. Rupture into the intestine also may occur. Rupture into the vagina and into the rectum has been observed in cases of tuberculosis of the bladder.

Prognosis.—Spontaneous recovery from chronic tuberculosis of the urinary organs is possible, as has been shown by personal observation, but occurs with extreme rarity, so that the prognosis of the disease is grave.

Treatment.—Persons with chronic tuberculosis of the urinary organs require, like cases of pulmonary tuberculosis, **fresh air and a nutritious diet.** If but one kidney is the seat of tuberculosis, it will be best to remove the diseased viscus from the body by operation, namely, to perform **nephrotomy** or **transverse resection of the kidney,** as, if delay be protracted, there will be danger of the disease extending to the ureter and the bladder, and possibly of involving the other kidney also. If both kidneys are diseased, *nephrotomy* will frequently have to be sufficient, if considerable portions of the kidney are still unattacked, in order to preserve these for the secretion of urine. Surgical measures also have been proposed in the treatment of tuberculosis of the bladder, and at times **scraping—curettement**—has been resorted to, and at other times **cauterization** of tuberculous ulcers.

Not much will be accomplished with **internal remedies** in the treatment of tuberculosis of the urinary organs. *Lime-water* in milk (1 tablespoonful to a cup of milk twice or thrice daily) has been administered in order to restrict the suppuration, and *astringents* also have been used. In a number of instances we have employed *boric acid* (2.0 : 200; 15 c.c.—1 tablespoonful—four times daily), although the results were uncertain. If the vesical symptoms are severe, daily *irrigation of the bladder* with dilute solutions of tannic acid (2.0–10.0 : 500) or silver nitrate (0.2–2.0 : 500) occasionally yield relief.

GENERAL MILIARY TUBERCULOSIS.

Etiology.—General miliary tuberculosis is invariably a *secondary* and *embolic infectious disease.* The process develops from the entrance of tubercle-bacilli into the blood-vessels or the lymphatics from a previously tuberculous organ, the bacteria being then carried to various viscera, and wherever they lodge and undergo multiplication they give rise to an irruption of miliary tubercles. Not rarely apparently healthy individuals are suddenly seized with general miliary tuberculosis. The disease may at first make the impression of being an independent disorder, but on post-mortem examination tuberculous-caseous foci will be encountered in one or another viscus, and which escape detection during life in consequence of their concealed situation. Acute miliary tuberculosis is frequently dependent upon *tuberculous caseation of the tracheobronchial lymph-glands*, and it has frequently been possible to demonstrate rupture of such glands into the pulmonary vessels. Only rarely have observations been recorded in which it has not been possible to detect the source of infection for general miliary tuberculosis, só that the disease did not appear to be a secondary disorder.

Generally infection is favored, as experience has shown, by certain circumstances—so-called *contributory factors*. Among these are *traumatisms*. Surgeons have frequently observed that general miliary tuberculosis has rapidly developed after operative treatment of tuberculous foci. An injurious influence is attributed also to *emotional disturbances*. Whether also the *season of the year* is of significance, as has been maintained, may be considered doubtful.

General miliary tuberculosis may occur at any age. In children it often develops in connection with chronic tuberculosis of the lymph-glands—scrofulosis.

Anatomic Alterations.—In cases of extensive miliary tuberculosis scarcely any organ remains free from miliary tubercles, which appear in small number even upon the endocardium and in the myocardium. In the **lungs** they can often be felt as innumerable hard nodules, like a bag filled with fine shot, and on section they project above the surface as pearl-gray, translucent nodules, often scarcely as large as the head of a pin. Not rarely they are visible through the pleura in innumerable amount. The **spleen** is generally enlarged and softened. Often it is exceedingly difficult to distinguish miliary tubercles from the follicles of the spleen. Diagnostic significance has been attached to the fact that miliary tubercles can generally be readily picked out of the splenic tissue with the point of a knife. In the **kidney** and the **liver** tubercles often appear as grayish or yellowish round dots with ill-defined boundaries. Frequently the great **omentum** is the seat of innumerable miliary tubercles, which can be seen with especial distinctness if the omentum be spread out and held toward the light. We shall refrain from a consideration in detail of miliary tuberculosis in other viscera, for any viscus may be the seat of the disease. Even the pancreas and the parotid glands, which formerly were thought invariably to be exempt, are now and again the seat of miliary tubercles. From a clinical-diagnostic point of view miliary tubercles of the *choroid* especially are of significance.

Symptoms and Diagnosis.—In the clinical picture of general miliary tuberculosis sometimes the profound **general infection,**

and at other times **local disease** of one or another viscus, predominates. I have had under observation individuals with general miliary tuberculosis who had been found unconscious in their rooms after having been able but a few hours previously to continue their pursuits in apparently unimpaired health. The patients exhibited subnormal temperature, presented a markedly cyanotic appearance, failed to return to consciousness, and died within from five to seven days without any organic change having developed. The suspicion of an intoxication had been awakened, but post-mortem examination disclosed extensive miliary tuberculosis.

In other cases general miliary tuberculosis is attended with high **fever**, and the clinical picture is suggestive especially of typhoid fever or septicemia. In some patients under my care there existed not only, as in typhoid fever, a dry, fissured, red tongue, meteorism, roseolæ, enlargement of the spleen, and diarrhea, but even fatal intestinal hemorrhage occurred, which, as the autopsy disclosed, resulted from tuberculous ulceration of the intestine. Naturally at the present day, it is true, the differential diagnosis would be possible with certainty from the presence of the blood-serum reaction of Widal. In contradistinction from septicopyemia significance is to be attached especially to examination of the eye-ground, for in cases of septic disease hemorrhage frequently occurs in the fundus of the eye, while in cases of miliary tuberculosis yellowish, circular spots—choroidal tubercles—not rarely make their appearance in the eye-ground. Hemorrhages into the external integument, as well as roseolous and erythematous lesions of the skin, would also be indicative of septic disease.

Occasionally the clinical course of general miliary tuberculosis resembles that of **intermittent fever,** chills, fever of several hours' duration, sweating and defervescence alternating at regular intervals. Progressive pallor and enlargement of the spleen likewise appear to indicate intermittent fever; but quinin, which is such a certain remedy in the treatment of intermittent fever, proves inefficacious, and, above all, malarial plasmodia cannot be found in the blood.

At times general miliary tuberculosis is concealed behind the clinical picture of **febrile bronchial catarrh.** The conditions must be considered suspicious if the temperature rises above 39° C. (102.2° F.) and is unyielding; and if, in addition, marked cyanosis is present.

General miliary tuberculosis may readily be confounded also with **fibrinous pneumonia.** High fever, symptoms of acute infiltration of one or several pulmonary lobes, sudden onset of the disorder, and rusty sputum scarcely permit of any doubt as to the accuracy of the diagnosis of fibrinous pneumonia. On post-mortem examination, although the lesions of an asthenic fibrinous pneumonia are encountered, it will at the same time be recognized that these are associated with general miliary tuberculosis of the lungs.

It should not be forgotten that often *tuberculous meningitis, pericarditis, pleuritis,* and *peritonitis* are only sequels of general miliary tuberculosis.

The presence of choroidal tubercles in the eye-ground is of especial significance in the recognition of general miliary tuberculosis, but these generally occur only when the disease is widely disseminated. Under such conditions a single examination of the eye-ground will not be sufficient. It is necessary to make ophthalmoscopic examinations frequently on successive days, as choroidal tubercles occasionally develop in the course of a few hours. At times it is possible to detect *tubercle-bacilli in the blood or the urine,* but especially in examining the blood great patience must be exhibited, and often many specimens must be prepared before even a single tubercle-bacillus is encountered. The procedure of puncturing the spleen with a sterilized hypodermic syringe, for the purpose of obtaining a small amount of *splenic juice,* and examining this for tubercle-bacilli, is not to be recommended on account of the attendant danger.

The *course of general miliary tuberculosis* may be acute, subacute, or chronic. In the last-named event the disease occasionally may persist for more than six or eight weeks, with a number of exacerbations and remissions, but it generally terminates fatally amid progressive exhaustion.

Prognosis.—General miliary tuberculosis is an almost certainly fatal disorder. Recovery is reported to have occurred in isolated instances, but all of the reports do not seem to be reliable.

Treatment.—No *specific treatment* for acute miliary tuberculosis is known, so that it is necessary to treat especially prominent symptoms in the usual manner.

TUBERCULOUS CEREBROSPINAL MENINGITIS.

Etiology.—Tuberculous meningitis is generally an associated manifestation of a more or less widely disseminated general miliary tuberculosis. The meninges are but rarely the seat of miliary tuberculosis in a predominant degree, but even under such circumstances the condition is one of secondary *embolic infection* of the meninges, arising from some other viscus the seat of tuberculosis. If it be impossible to detect a tuberculous focus during life, the tracheobronchial lymphatic glands are with especial frequency found, on post-mortem examination, to be tuberculous and cheesy. In children, in whom tuberculous meningitis occurs frequently, inflammatory enlargement of the tracheobronchial lymphatic glands occurs not rarely as a complication of antecedent whooping-cough, measles, or scarlet fever, with subsequent infection by tubercle-bacilli. Among one hundred autopsies I have encountered but two cases in which, in spite of most careful examination of the body, a tuberculous-cheesy focus could not be detected; and as tubercle-bacilli have been found also in the nasal mucus of healthy individuals, the occurrence of *primary tuberculous meningitis* cannot be wholly excluded, inasmuch as tubercle-bacilli may gain entrance into the interior of the skull from the nasal mucous membrane. Under any circumstances the condition would be an exceptional one. Occasionally the occurrence of tuberculous meningitis has been preceded by the injurious influences that were mentioned as *contributory factors* for embolic infection in cases of general miliary tuberculosis, as, for instance, exposure to cold, traumatism, and excessive mental activity.

Anatomic Alterations.—After removal of the bony calvarium the marked **tension of the dura mater** is generally conspicuous. The surface of the **cerebral convolutions** appears flattened and the **sulci** obliterated, as if the brain had been compressed with some violence against the inner surface of the skull. The **veins of the pia** are generally distended with blood, and the entire surface of the brain is greatly reddened. After removal of the brain isolated translucent, pearly-gray tubercles upon the dura can usually be recognized, particularly upon the clivus and in the occipital fossa, as well as in the area of distribution of the middle cerebral artery. The **sinuses of the dura** generally contain blood and coagula in abundance. The principal alterations are present in the **pia** and the **arachnoid,** which exhibit a turbid appearance and are infiltrated with a fibrinous-serous, less commonly a slightly purulent exudate, particularly in the interval between the pons, the cerebral peduncles, and the optic chiasm. The development of tubercles is likely to be particularly active in the course of the **Sylvian artery,** so that it is well, in cases in which miliary tubercles cannot be distinctly recognized in other situations, to scrutinize this situation.

On opening the *cerebral ventricles* these will be found to be more or less greatly dilated and filled with a large amount generally of slightly turbid and flocculent fluid, so that *acute internal hydrocephalus* exists. Often the **wall of the ventricle** is partly softened—white cadaveric softening—and the **foramen of Monro** is enlarged beyond the size of a pea. Also miliary tubercles are not rarely to be found in the ependyma of the ventricles and in the **choroid plexus.** The **cerebellum** also is likely to exhibit a gelatinous, serous infiltration in the median line upon its superior surface.

The **spinal cord** is almost unexceptionally involved in tuberculous meningitis. Occasionally only miliary tubercles can be found in the soft membranes of the cord, but often a gelatinous-serous exudation and infiltration have also taken place, so that the pia appears thickened and whitish or greenish. These alterations are developed principally upon the dorsal aspect, probably in consequence of the persistent dorsal decubitus, and frequently almost avoid the cervical portion of the cord. The remaining viscera generally contain miliary tubercles in greater or lesser number.

On *microscopic examination* it will be found that the formation of tubercles begins in the walls of the vessels. The tissues of the meninges are, besides, abundantly infiltrated with round cells. Not rarely the formation of tubercles and cellular infiltration extend into the structure of the brain and the cord along the pial sheaths.

Symptoms.—Tuberculous meningitis is preceded with exceeding frequency by **premonitory symptoms** or prodromes of extremely indefinite character for several days or even weeks. The patient feels languid, complains frequently of distressing headache, is sleepless, loses appetite, and occasionally vomits repeatedly. The conditions become materially more serious if **inequality of the pupils** is superadded; and scarcely any doubt as to the diagnosis will remain if **rigidity of the neck** develops, which usually persists throughout the entire subsequent course of the disease, and frequently is likely to disappear only in profoundly comatose patients a short time before death. Percussion of the skull and the vertebral column is generally attended with tenderness, and is capable of exciting manifestations of pain even in unconscious patients. Progressive **coma** soon sets in. The patients frequently emit a sudden, short, and piercing cry—the **meningitic** or **hydrocephalic cry**. **Cutaneous hyperesthesia** is generally present, so that even slight pinching of the skin causes severe pain. The *cutaneous vasomotor nerves* are unduly irritable; after mechanical irritation of the skin, as, for instance, rubbing a hard substance over the surface, redness will persist for some time—**meningitic spots.** The **knee-jerks** are variable, at times exaggerated, at other times diminished or lost. I have often observed toward the close of the disease disappearance of previously exaggerated knee-jerks.

The **bodily temperature** generally exhibits a variable course. In the majority of cases fever is present, but cases also occur without a febrile course, even with subnormal temperature. Such fever as is present pursues a wholly irregular course. Temperatures above 40° C. (104° F.) are, however, rare.

The **pulse** often exhibits important alterations. At the beginning of the disease frequently it is unusually slow in consequence of irritation of the vagus. In the further course of the disease, however, the pulse becomes gradually more frequent, and, finally, can occasionally be scarcely counted, because the stage of irri-tion has been followed by paralysis of the vagus. Naturally grave prognostic significance will be attached to acceleration of the pulse.

Changes in **respiratory movement** occur with great frequency. The breathing may assume the Cheyne-Stokes character, the respiratory movements slowly increasing and diminishing, and intermissions in respiration (apnea) occurring, which are then followed again by increased and diminished frequency of breathing. The occurrence of Cheyne-Stokes breathing is attributed to innervational disturbances in the respiratory and vasomotor centers in the medulla oblongata, and these are due in part to increased intracerebral pressure, and in part to circulatory alterations in the medulla. Biot's breathing occurs less commonly. It is characterized by intermissions in breathing, which are followed by regular respiratory movements.

The **abdomen** is generally scaphoid or trough-shaped, so that the pulsation of the abdominal aorta can be felt beneath the abdominal walls, which, at the same time, are generally distended with board-like hardness, and for this reason the retraction of the abdomen is probably attributable to persistent contraction of the abdominal muscles. There is probably, also, spasm of the muscular coat of the bowel, which explains the obstinate **constipation** usually present. Often repeated **vomiting** occurs in consequence of irritation of the vagus.

The *course* of the disease covers from a few days to a few weeks; occasionally the duration is longer than two months. As a rule, death occurs amid progressive coma in consequence of cardiac or cerebral paralysis. In cases that pursue a chronic course periods of remarkable improvement occasionally occur, so that unconscious patients are restored to consciousness, and they may even get out of bed ; but one should not be deceived by such occurrences, for almost unexceptionally a relapse does not fail to take place, and death occurs finally unchecked.

Among the *complications muscular spasm* and *paralysis* should be mentioned first. Clonic spasm occurs at times in certain groups of muscles, while at other times it takes place in certain members, although it may appear in unilateral or in general distribution. If the muscles of mastication are involved, *grinding of the teeth* will result, a symptom that is frequently present in cases of tuberculous meningitis. Muscular paralysis also occasionally involves the distribution of a single nerve, or monoplegia or hemiplegia may occur. Further, it is by no means always possible on post-mortem examination to discover an anatomic basis for the paralysis.

Among the *anomalies of tuberculous meningitis* the *apoplectiform variety* especially should be mentioned. This sets in suddenly, with loss of consciousness, and simulates the clinical picture of cerebral hemorrhage. This resemblance becomes the greater if paralysis appears. I have, under such circumstances, observed right hemiplegia and aphasia, and have found on post-mortem examination that especially the distribution of the right Sylvian artery was the seat of large numbers of tubercles, and of gelatinous infiltration and inflammation in particularly marked degree.

Diagnosis.—In the majority of cases tuberculous meningitis can be readily recognized. Difficulties arise, as a rule, only at the beginning of the disease, so long as rigidity of the neck, headache, and coma are not present. In addition, an effort should be made to determine the source of origin for the meningeal infection, and, among other conditions, examination of the epididymis, the prostate, and the seminal vesicles for tuberculous foci, and of the ear for tuberculous caries of the petrous bone, should not be omitted.

In contradistinction from **purulent meningitis** tuberculous meningitis is characterized by its more insidious onset and course, and the absence or the slight character of the febrile movement. In the diagnosis of tuberculous meningitis the presence of *choroidal tubercles* is of especial significance; but this occurs only in about one-sixth of the cases, and is to be expected only if, in addition to the meninges, many other organs are the seat of miliary tubercles. Under such circumstances it should not be forgotten that it is necessary to examine the patient from day to day for miliary tuberculosis of the choroid, as this may develop in the course of a few hours. Not rarely *lumbar puncture* will aid in reaching a correct diagnosis. The fluid obtained on puncture is, in contradistinction from that obtained in cases of purulent meningitis, in which it is generally turbid and flocculent, and purulent in appearance, usually clear, and on standing a thin, gray cloud forms, in which in the majority of cases tubercle-bacilli can be demonstrated by the usual staining-methods. It should further be recalled that occasionally tubercle-bacilli can be found in the blood or the splenic juice.

Prognosis.—The prognosis of tuberculous meningitis is unfavorable. Quite rarely, contrary to the rule, recovery has been observed, but in some instances this appears to be questionable.

Treatment.—The treatment of tuberculous meningitis agrees wholly with that described on pp. 447, 448 for purulent meningitis.

TUBERCULOUS PERITONITIS.

Etiology.—Tuberculous peritonitis is frequently associated with **intestinal tuberculosis** or **tuberculosis of the abdominal lymphatic glands.** It occurs, therefore, not rarely in children in consequence of the use of milk derived from tuberculous cows. In women **tuberculosis of the oviducts or the ovaries** is frequently the source of origin for secondary infection of the peritoneum. In any event the disorder is almost unexceptionally *secondary* in origin, and it is generally preceded by tuberculosis in other organs.

Symptoms.—Tuberculous peritonitis is often attended with the formation of a **fluid exudate**, which is generally serous, less commonly hemorrhagic, or even purulent, putrid, or fatty. The patient complains principally of slowly progressive **increase in the size of the abdomen**, which develops either wholly without pain or with slight cutting and griping sensations. The greater the enlargement of the abdomen the more marked become the *sense of constriction and the dyspnea*. Febrile movement is wanting, or occurs irregularly, and is almost always slight. Examination of the abdomen discloses a *free effusion into the abdominal cavity*. Fluctuation can therefore be elicited, and with the patient in the dorsal decubitus a loud tympanitic note over the anterior portion of the abdominal wall, and a dull percussion-note on the sides. On change of posture to the lateral decubitus, however, the dulness disappears from the now uppermost lateral aspect of the abdomen, and is replaced by a tympanitic note. Occasionally the abdominal walls are tender to touch in places. At times a transversely situated, hard, nodular, cord-like body, corresponding to the *great omentum* thickened by tubercles, and generally also contracted, can be felt beneath the anterior abdominal wall at about the level of the umbilicus. Occasionally a slight inflammatory infiltration of the skin becomes noticeable at the umbilicus, but this is not at all a frequent, nor even a constant, occurrence, as it has been stated to be.

The *course of tuberculous serous peritonitis* is generally chronic, occasionally being protracted for more than a year. Often periods of absorption and increased accumulation of the peritoneal fluid alternate repeatedly with each other. The greater the degree in which the fluid disappears from the abdominal cavity the more distinctly palpable does the thickened and shrunken omentum become. Even after complete absorption of the fluid *relapses* frequently occur, and these may occasionally not set in until the lapse of from one to three years. Reaccumulation of fluid will fail to take place only if complete *obliteration of the peritoneal cavity* has resulted in consequence of adhesions. Occasionally the recovery is but incomplete, the peritoneal cavity being obliterated in places, while in other situations fluid exudate remains in circumscribed and encapsulated cavities.

A fatal *termination* is not rare. At times this occurs in consequence of *suffocation* resulting from marked displacement upward of the diaphragm, the lungs, and the heart, in the presence of an excessive accumulation of fluid. In some cases death is due to excessive *debility*, although it may result also from general *miliary tuberculosis* or from the *primary disorder*, as, for instance, intestinal tuberculosis.

A rather uncommon variety of tuberculous peritonitis is unattended with the accumulation of a fluid exudate. In this condition tuberculous-cheesy thickening occurs, principally in the great omentum, which occasionally is confounded with an abdominal tumor. Under such circumstances palpable and audible *peritonitic friction-phenomena* can also be appreciated.

Diagnosis.—The diagnosis of serous tuberculous peritonitis is not always easy. The disorder may especially be confounded with **chronic serous non-tuberculous peritonitis,** which is occasionally observed in youthful persons, particularly young girls at the period of puberty. It is noteworthy that even after abdominal section error in diagnosis is not impossible, because there occurs a **chronic fibromatous serous peritonitis,** in which the peritoneum is covered with innumerable small, connective-tissue nodules, which macroscopically wholly resemble tubercles. Only microscopic examination is decisive in the differential diagnosis.

Formerly tuberculous peritonitis also was probably considered as *idiopathic ascites,* although inflammatory fluid is distinguished from a transudate by its higher specific gravity (above 1015), and the presence of a larger amount of albumin (above 0.4 per cent.). At times confusion with **cirrhosis of the liver or thrombosis of the portal vein** occurs. Under such circumstances icteric manifestations should be looked for.

Prognosis.—The prognosis of tuberculous peritonitis is grave. Permanent recovery occurs but seldom. As a rule, recovery is, at best, but temporary, and is followed in the course of a few days at the latest by exacerbations, which terminate fatally.

Anatomic Alterations.—The distinctive anatomic alterations consist in the development of tubercles upon the peritoneum. These are likely to be especially numerous in the great omentum, so that this structure may attain the thickness of a finger. Occasionally the great omentum is transformed into a club-shaped, multinodular, and in part cheesy, body as thick as an arm, which passes transversely across the middle of the abdomen. The peritoneum itself also is not rarely the seat of diffuse, in part cheesy, thick infiltrates. Tubercles are often surrounded in places by extravasations of blood of varying age, so that some are bounded only by a black ring of pigment. Adhesions between the intestines and between the great omentum and the abdominal wall, as well as between the liver and the spleen, and the diaphragm, are quite frequent.

Treatment.—In the treatment of tuberculous serous peritonitis, the results of **abdominal section—celiotomy—**have attracted considerable attention, as failure of the fluid exudate to reaccumulate and the occurrence of temporary recovery have frequently been observed to follow. Reaccumulation of the fluid is naturally impossible if, after abdominal section, all of the fluid has been evacuated and total obliteration of the peritoneal cavity has resulted. More extensive experience has shown that the results of surgical treatment for tuberculous peritonitis are by no means so brilliant as they at first appeared to be. Reaccumulation of the fluid exudate occurs by no means rarely, or the tuberculous process continues to progress and eventually leads to a fatal termination, in spite of failure of the fluid to reaccumulate. I have had under observation a small number of patients who were operated upon without success, and whom I was able to relieve of the effusion by means of internal or derivative treatment.

Recently I have made frequent use especially of **inunction of the abdominal walls with green soap.** I had the abdominal walls rubbed once daily with green soap until active redness and slight inflammation of the abdominal integument developed. Then the inunctions were intermitted until the inflammatory phenomena had disappeared, and the procedure was constantly repeated until absorption of the fluid exudate had been completed.

In some patients I have secured considerable diuresis through the administration of large doses of **calomel** (0.5—7½ grains—thrice daily), in the sequence of which the abdominal fluid disappeared. Diuretin exerted as little influence upon the secretion of urine, in cases under my care, as digitalis and urea. On the other hand, some patients were relieved of their exudate by active diaphoresis **in a sweat-chamber.**

TUBERCULOUS PLEURITIS AND PERICARDITIS.

Tuberculous pleuritis and pericarditis are generally *secondary* in origin. They occur with especial frequency in the course of chronic tuberculosis of the lungs or the tracheobronchial lymphatic glands. The symptoms are those of fibrinous or exudative pleuritis and pericarditis respectively, and it is often extremely difficult to recognize the tuberculous character of the inflammatory process. Most cases of spontaneous serous pleuritis or pericarditis are tuberculous in origin. Occasionally the exudate is hemorrhagic, purulent or putrid, even fatty. That pleuritis or pericarditis is tuberculous can be most certainly recognized by withdrawing some of the exudate with a syringe, injecting it into the abdominal cavity of guinea-pigs, and noting, after the lapse of from four to six weeks, whether tuberculosis has developed in the inoculated animals.

The tubercles at times cover the serous membranes in innumerable amount, while at other times they constitute confluent infiltrates, frequently cheesy in places, and occasionally in process of disintegration. If adhesions have formed between the two serous surfaces, these are with especial frequency covered with numerous tubercles. Occasionally tuberculous inflammation of all of the serous membranes, especially the pleura, the pericardium, and the peritoneum, has developed simultaneously, both clinically and anatomically, and this condition has, by some clinicians, been designated tuberculous serositis. Tuberculous pleuritis and pericarditis may undergo recovery, but the cure is generally not permanent, and within a shorter or longer period the inflammatory process recurs or tuberculosis develops in other organs.

The **prognosis** accordingly is not favorable.

The **treatment** is the same as that for non-tuberculous pericarditis and pleuritis.

SCROFULOSIS.

Etiology.—Scrofulosis is an exceedingly widespread *disease of childhood,* in which *chronic tuberculosis of the lymphatic glands* constitutes the most conspicuous manifestation. In addition, tuberculous lesions often occur also in other organs, with especial frequency in the bones and joints; and, besides, a marked tendency to chronic inflammatory processes of the most varied kind is noticeable.

The *predisposition to scrofulosis* is either *congenital* or *acquired.* Experience has shown that scrofulosis occurs particularly in children whose parents marry late in life, or if the parents were suffering from carcinoma, tertiary syphilis, tuberculosis, or other debilitating disease at the time of conception. The *character of the food* and the *mode of life* have a great influence upon the development of scrofulosis. The use of excessive amounts of amylaceous food, particularly bread, potatoes, and farinaceous articles, favors the occurrence of scrofulosis, and the same is true of a deficiency of fresh air. Scrofulosis has even been observed to develop in adults who have for a long time been deprived of fresh air, as, for instance, in prisoners—prison-scrofulosis. Not rarely the first symptoms of scrofulosis appear after recovery from the *diseases of childhood,* as, for instance, measles, scarlet fever, chicken-pox, or whooping-cough, obviously because the body has been debilitated and has offered less resistance to the invasion of tubercle-bacilli.

In what manner and from what source tubercle-bacilli gain entrance into the lymphatic glands is not known. Inhalation, perhaps in still greater degree infection through the use of milk from tuberculous cows, will principally have to be thought of.

Symptoms, Diagnosis, and Prognosis.—*Scrofulous enlargement of the lymphatic glands* frequently begins in the first months of life. The submaxillary and cervical glands are usually involved earliest, but not rarely internal lymphatic glands also participate in the alterations, particularly those of the anterior mediastinum and the abdominal cavity. Below the lower jaw and along the neck not only can the enlarged glands be felt as multiple, roundish, and indurated tumors, but they can often also be seen, as they may attain the size of a hen's egg. Generally they are insensitive to pressure. The overlying skin appears unaltered and immovable. Enlargement of the mediastinal lymphatic glands will occasionally be indicated by dulness on percussion over the manubrium of the sternum, and enlarged abdominal lymphatic glands are sometimes palpable through the abdominal walls as multinodular tumors.

Not rarely **softening** and **inflammatory alterations** occur, especially in the submaxillary and cervical lymphatic glands. Individual glands become sensitive to touch, progressively softer, and eventually distinctly fluctuating. As a result peri-adenitic adhesions to the external skin take place. This becomes reddened and infiltrated, and finally pus, generally of thin consistence, ruptures through the external skin. There then often remains a fistula, which secretes thin pus for many months, and if it heals is likely to leave a retracted, multiradiate cicatrix that persists throughout life, and is indicative of the existence of antecedent scrofulosis. Not infrequently contiguous lymphatic glands become involved in a similar manner after a time, and the subsequent cutaneous cicatrices situated close together cause considerable disfigurement in the cervical region. Further, it may also happen that the pus from the lymphatic glands does not rupture externally, but burrows downward beneath the skin, under some circumstances reaching the mediastinum, or the pleural or the pericardial cavity, where it gives rise to grave inflammatory complications. There is a possibility also that the large blood-vessels of the neck may be eroded by the pus, and fatal hemorrhage result in consequence.

Scrofulous individuals not rarely present a peculiar **facial expression**, so that the designation *scrofulous habitus* has been employed. The face frequently is plump in appearance, the lips being swollen and everted, and the nose acquiring a pear-like or potato-like shape. Often the children are also slow and phlegmatic, both in mental and in physical activity, and under such circumstances the designation *torpid scrofulosis* has been employed. In contradistinction from this an *erethetic scrofulosis* has been described. Children presenting this variety of the disease exhibit an almost abnormally increased activity of body and mind. The vasomotors also are in a state of excessive irritability, and a vivid redness of the skin appears even after slight excitement.

Not rarely the scrofulous changes are confined to the alterations in the lymphatic glands, but in other instances inflammatory *complications* are superadded, which may be either tuberculous, therefore specific, or non-tuberculous in character. Among the *tuberculous complications* there occur upon the external integument ***lupus***, and, perhaps, also *lichen of the scrofulous;* lupus occurs also upon the mucous membranes. The *bones* and the *joints* frequently become the seat of tuberculosis, and there result, in consequence, tuberculous caries of the bones and its sequelæ, or articular fungus, arthrocace. Occasionally the lungs or other viscera become the seat of tuberculosis, and the like.

Among the *non-specific complications*, obstinate *eczema* may be mentioned. The *tonsils* frequently are enlarged, so that speech and oronasal respiration are interfered with. Often *acute and chronic inflammation of the pharynx* occurs. Acute and chronic *nasal catarrh* also is a frequent symptom of scrofulosis. Scrofulous individuals exhibit an unmistakable tendency to chronic and acute *inflammation of the air-passages*. The organs of *special sense* not rarely suffer. In the *ear*, in addition to extremely obstinate eczema of the auricle and the external auditory canal, *tuberculous caries of the petrous bone*, with its serious dangers, is particularly to be mentioned. Scrofulous individuals suffer frequently from chronic disease of the *eyes*, such as blepharitis, blepharadenitis, conjunctivitis, keratitis, etc.

Occasionally remarkable alterations take place in the intestine that have been designated *mesenteric tabes*. The children suffer from almost uncontrollable diarrhea, and have an insatiable appetite, but, nevertheless, they emaciate progressively. The skin becomes loose and thrown into folds, and almost falls about the emaciated members. The face becomes sunken, its prominent portions project sharply forward, and the little patients acquire the markedly wrinkled facies of an old person. The abdomen is distended like that of a frog, and tense and drum-like. The intestinal evacuations are characterized by an almost putrid odor. The majority of the children die in consequence of exhaustion.

Scrofulosis generally pursues a most *chronic course*, and occasionally is protracted over many years. In addition to the complications already mentioned, it is attended with the further danger that it may unexpectedly be the source of origin for a general miliary tuberculosis. There is also a possibility of *amyloid degeneration* of the viscera developing. The prognosis, therefore, is always serious.

Anatomic Alterations.—Scrofulous lymphatic glands are conspicuous, in the first place, on account of their large size, which is due in part to inflammatory hyperplasia and in part to tuberculous proliferation. Tubercle-bacilli are present in the latter in but small number, and are generally found especially in such giant-cells as may be present. After the disease has existed for a considerable time caseation takes place, with desiccation and partial fatty degeneration of the proliferated cells, and this will be manifested by dryness and yellowish discoloration of the tissues. The glands upon section then not rarely present the appearance of a boiled potato. Although tubercle-bacilli are generally not found in the cheesy masses, these must contain spores of the tubercle-bacilli that are not appreciable to the eye, for they prove infectious in inoculation-experiments.

Treatment.—Considerable importance is to be attached to *prophylactic measures* in the *prevention of scrofulosis.* Children should be kept a good deal in the *open air;* but this rule is applicable, also, to adults, and recovery has been repeatedly observed in scrofulous individuals when they could be made to spend a considerable time daily out of doors. In addition, the *diet* will require consideration. For children during the first two years of life milk should be the principal article of food. Subsequently a mixed diet is to be recommended, but a preponderance of amylaceous food should be avoided, and greater importance is to be attached especially to the ingestion of meat and fresh vegetables.

In the presence, also, of actual scrofulosis the **diet** and the **mode of life** are of far greater importance than drugs. Among the latter **codliver-oil** is much used, but this would seem to be indicated only in cases of erethetic scrofulosis:

℞ Codliver-oil, 200.0 (6½ fluidounces).
Dose: 20 c.c. (⅔ fluidounce—1 tablespoonful) twice daily.

To avoid gastric derangement the codliver-oil should be given only during the cool season of the year, and its use should be intermitted for from two to four weeks every four weeks. Iron should be administered to pallid children, especially the *iodid;* as for instance:

℞ Sirup of iron iodid,
Simple sirup, each, 25.0 (⅚ fluidounce).—M.
Dose: 5 c.c. (75 minims) thrice daily.

Saline baths are much employed, and these may be prepared for poor patients by adding from two to five pounds of rock-salt to a full bath. The bath can be used repeatedly, most advantageously by removing a certain amount of water, and replacing it with warm water until the temperature of the bath reaches 35° C. (28° R.—95° F.), and, besides, adding a corresponding amount of salt. Other forms of salt bathing (Kreuznach, Strassfurt, Wittekind, Kösen, Rheinfelden, Schweizerhall, etc.) may be employed in a similar manner. If concentrated brine be employed for saline baths, from five to ten liters may be added to a full bath.

The children of well-to-do parents are often sent to *saline baths* during the summer, where, particularly if they live in large cities, they will have the further advantage of spending a great deal of time in the fresh air. Among such saline baths Kösen, Bex in the Canton of Waadt, Homburg, Kreuznach, Nauheim, Rehme-Oeynhausen, Reichenall, Rheinfelden, Salzschlirff, Salzungen, Schweizerhall, Soden, Sulza, and Wittekind may be mentioned.

Recently **sanatoria** have been erected at the seaside where poor children are in part received gratuitously, for the purpose of permitting the patients to spend a considerable length of time in the sea-air. Experience thus far indicates that improvement does not take place at the seaside in children suffering from inflammatory alterations (ostitis, arthritis, ophthalmitis, eczema, glandular suppuration). Local inflammatory processes should be treated in the usual manner.

SOLITARY TUBERCULOSIS.

Occasionally tuberculous hyperplasia gives rise to extensive cheesy or purulent and softened *tumors* that may become larger than a fist, and are designated *solitary tubercles.* They result from the coalescence of smaller tubercles, and occur especially in the brain and the spinal cord, but at times also in the heart-muscle, the liver, and the spleen. During life they occasion the symptoms of a tumor or an abscess, and often it is difficult, even at the autopsy, to determine their nature with certainty. At times they give rise to the development of miliary tuberculosis. Not rarely they are unattended with symptoms. The condition is generally one of *secondary tuberculosis.*

III. SYPHILIS.

Syphilis, also designated *lues*, is included, together with gonorrhea and chancroid, among the *venereal diseases.* While gonorrhea and chancroid, however, are purely local diseases of the sexual organs, syphilis is attended with *general infection.* From this circumstance it can be understood that the disease may be transmitted from syphilitic parents also to their offspring, so that a distinction is to be made between *acquired* and *inherited syphilis.* The *infectious agent* is not known. The disease first attracted general attention in December, 1494, from its occurrence on a large scale among the troops of Charles VIII. of Anjou, who at the time were occupying Naples. Probably, however, syphilis occurred even earlier, although it was encountered but seldom before this time.

ACQUIRED SYPHILIS.

ACQUIRED SYPHILIS IN THE PRIMARY AND THE SECONDARY STAGE.

Etiology.—Syphilitic infection results most frequently through sexual intercourse—*genital infection.* The *blood* of a syphilitic and the *secretions from syphilitic eruptions upon the skin and the* *mucous membranes—syphilids*—are infective, and among these the smeary accumulations upon broad condyloma are especially dangerous. If wounds are present upon the sexual organs, or if these be incurred during sexual intercourse, they may be readily inoculated with blood or infectious discharges. Further, it is not inconceivable that at times inoculation may take place through the hair-follicles in the absence of any lesion of the skin.

Physiologic secretions from syphilitic individuals contain no infective matter, and the saliva, the nasal and the lacrimal secretion, the secretions from the air-passages, the milk, and the urine become infectious only when admixed with blood or discharges from broad condylomata. The *spermatozoids* and the *ova* of syphilitic individuals are, however, infected, and this fact explains the hereditary transmission of the disease.

Like gonorrhea and chancroid, syphilis occurs most frequently in *unmarried individuals between the twentieth and the fortieth year* of life. It is acquired with especial frequency in *houses of prostitution* subject to inadequate or to no medical supervision. Should prostitutes suffering from syphilis reside for no longer than a week in such houses, receiving many male visitors, they may during this period inoculate more than a hundred men. Should syphilis occur through genital infection in *children* defloration will always have to be considered.

Extragenital—accidental—infection with syphilis may result from *kissing*, for not rarely broad condylomata are situated upon the lips of syphilitic individuals, so that transference of syphilitic secretion may take place to fissures upon the lips of healthy individuals. Syphilitic infection is occasionally conveyed through *domestic utensils* (glasses, pipes, cigar-holders, cigars, etc.), because syphilitic secretion often adheres to these. Transmission of syphilis through *surgical instruments* that have been employed in operations upon syphilitic patients, and subsequently in operations upon healthy individuals without previous thorough sterilization, scarcely occurs at the present day, because all instruments are, as a rule, thoroughly sterilized before and after being used. Occasionally *physicians, midwives*, or *nurses* with sore fingers acquire syphilitic infection as a result of examining or nursing syphilitic patients. Conversely, however, syphilis may be conveyed to healthy individuals through syphilitic physicians or midwives, and by the latter, particularly, if they indulge in the vicious and antiquated practice of moistening the umbilical wound with their saliva.

Syphilis has occasionally been transmitted to children through the rite of *circumcision* when the operator was syphilitic, because according to the Mosaic religion the hemorrhage should be controlled by sucking the wound.

Syphilis has been observed in the past in isolated instances as a result of *vaccination*, when the virus was obtained from a syphilitic source, and the lymph employed contained, in contravention of proper precautions, blood visible macroscopically. As at the present day calves' lymph—animal lymph—is employed for vaccination, the occurrence of vaccinal syphilis is excluded. Occasionally syphilis has been observed after ***bite-wounds*** or ***scratch-wounds*** inflicted by syphilitic individuals.

Syphilis, unfortunately, is an *exceedingly widespread disease*. In general, it is encountered more frequently in *large cities*, particularly in seaport cities, than in the country, because in the former the tendency to excesses of all kind, and the opportunities for their indulgence, are more common. The majority of persons are attacked by syphilis but once. Repeated infection, or *reinfection*, occurs with extreme rarity.

Symptoms.—The *period of incubation for syphilis* is about four weeks. The first appreciable symptoms are the **hard chancre** and **multiple indolent buboes** of the nearest adjacent lymph-glands. The *hard chancre* can be readily recognized. It consists in a sharply circumscribed nodule of cartilaginous hardness, which is scarcely sensitive to pressure, and does not bleed when compressed. The overlying skin is often reddened and peculiarly smooth and glistening; slight desquamation of the epidermis also not rarely takes place. The nodule varies in size from barely that of a lentil to five centimeters in diameter, and rarely beyond.

In some cases the hard chancre acquires but slight thickness, so that on compression between the fingers it feels almost like a sheet of cardboard. For this reason the designation *parchment-chancre—papyraceous* or *foliaceous hard chancre*—has been employed. Such a chancre will at times be situated in fissures and folds of the skin, and may then readily escape observation.

If a hard chancre has existed for some time (from four to eight weeks), involution slowly takes place, usually beginning in the middle, and being attended with gradually increasing excavation. Generally, indurated cicatricial thickening remains for a long time at the site of the chancre, and this at times develops into a new nodule without especial provocation—*recurrent hard chancre*.

On *microscopic examination of a hard chancre* the *cutis* will be found *densely infiltrated with round cells*, which in part have invaded the epidermis. In addition, *changes are present in the blood-vessels and the lymphatics*. The walls of the blood-vessels are thickened and infiltrated with round cells, and, at the same time, obliterating endarteritis has developed, giving rise to constriction, and here and there to occlusion, of the lumen of the vessels. The lymphatics usually are greatly dilated. Their endothelial cells appear to be in a state of proliferation and of partial desquamation.

Among the *complications of hard chancre ulcerative destruction* may be mentioned. This at times involves only the most superficial layers, but at other times it extends deeply, and gives rise to a sharply circumscribed, deep ulcer, that is occasionally covered with necrotic masses. If the hard chancre be situated upon the prepuce or in the coronary sulcus, it may readily give rise to *phimosis*, *paraphimosis*, or *balanoposthitis*.

The *seat of a hard chancre* is in the male most frequently the prepuce, and in the female the greater labia, occasionally the cervix of the uterus. In women it is often exceedingly difficult to demonstrate the presence of a hard chancre, because this frequently soon undergoes softening, and then it resembles a broad condyloma. Occasionally a hard chancre is situated in the anterior portion of the urethra—*concealed hard chancre.* Under such circumstances there is generally a purulent discharge from the urethra, which may be readily mistaken for gonorrhea, although gonococci are not present, and, besides, the hard chancre can generally be felt from without as an indurated nodule. In cases of extragenital infection the hard chancre may be situated in all possible portions of the body, as, for instance, in physicians on the examining index-finger. Hard chancre occurs with relative frequency upon the lips. Occasionally it is encountered upon the tonsils. Not rarely the mouth has, under such circumstances, been used in unnatural practices. The recognition of a hard chancre in portions of the body other than the genitalia is occasionally attended with difficulty. Under such circumstances great weight is to be attached to enlargement of the adjacent lymphatic glands.

With the appearance of a hard chancre *multiple indolent buboes* in the adjacent lymphatic glands are associated. If the chancre be situated upon the genitalia, the inguinal lymphatic glands on both sides become enlarged; if the chancre be situated upon the finger, the adjacent glands at the elbow, occasionally, however, also the axillary glands, are affected; and if the chancre be situated upon the lips, the submental or the submaxillary lymphatic glands are enlarged.

In the presence of inguinal buboes a number of round bodies, averaging in size that of a cherry, can be felt beneath the skin; these can be distinctly differentiated from each other, are covered by skin that is unchanged and movable, and are insensitive to pressure. Occasionally also the lymphatics upon the dorsum of the penis are, in consequence of lymphangitis, converted into hard, cylindrical cords, in places exhibiting somewhat nodular enlargement.

So long as the symptoms of syphilis are restricted to the hard chancre and buboes in the adjacent lymphatic glands the condition is included in the *first stage of syphilis* or the *stage of the initial sclerosis.* A period of six weeks then generally elapses, during which the patient exhibits no material alteration. This interval has been designated also the *second period of incubation of syphilis.* Often the patient becomes paler and paler. Above all, however, **general enlargement of lymphatic glands** gradually takes place, probably as an evidence that the syphilitic virus has been slowly distributed throughout the entire body. Painless enlargement of the epitrochlear and occipital lymphatic glands especially is indicative of a syphilitic glandular involvement. The **spleen**, also, is frequently increased distinctly in size.

The *secondary stage of syphilis* begins about the eleventh week after antecedent infection, and this may be designated also the *stage of syphilids of the skin and the mucous membranes*, or the *condylomatous stage*. Not rarely this begins with chilliness and fever, and there is danger, on careless investigation, of confounding syphilis with acute infectious diseases, particularly measles and typhoid fever. Often the patients complain of severe pain in the muscles and fasciæ; particularly, however, in the bones. In the latter situation the pains are principally increased at night. Often they are situated in the cranium or the tibia, and inflammatory thickening of the periosteum is occasionally found in these situations. The bone-pains have been designated osteocopic or boring pains.

The cutaneous alterations—**syphilitic exanthemata** or **syphilids**—are dependent at times upon hyperemic, at other times upon papular, squamous, vesicular, or pustular processes in the skin. The most frequent syphilid is the *syphilitic roseola*, which occurs as roundish, brownish-red spots, disappearing on pressure with the finger. These are especially abundant upon the trunk, but they occur also upon the extremities and the face, in the latter situation particularly at the junction of the forehead with the scalp. Less commonly than the roseola, diffuse redness of the skin appears—*syphilitic erythema*—whose appearance is suggestive of scarlet fever.

Among the papular syphilids the *broad condyloma* occupies the first place by reason of its frequency and its importance. At the height of its development this appears as a flat, broad elevation of the skin, covered with a smeary, gray, rancid deposit of offensive odor, but possessing exceedingly infective properties. Broad condylomata are circular, often oval in outline, and vary in size from a few millimeters to several centimeters in diameter. They develop with especial frequency upon parts of the body where cutaneous surfaces come in contact, as, for instance, upon the inferior surface of the penis and the adjacent skin of the scrotum, between the scrotum and the surface of the thighs, between the labia, surrounding the anus, in the inguinal folds, in the umbilical depression, in the axillary cavity, between the nasal alæ and the skin of the cheek, at the angles of the mouth, and between the fingers and the toes. In the last-named situations they frequently undergo disintegration, and ulcers in consequence form between some of the toes, and these are strongly indicative of a syphilitic origin. Often broad condylomata are grouped closely together, and frequently they can be distinctly seen to have given rise to an exact reproduction on the opposite surface, obviously through auto-infection. If the *development of broad condylomata* be more carefully studied, it will be found that they begin as dry, generally reddened papules upon the skin, and that the smeary deposit upon them only forms gradually through moistening and maceration of the epidermis. Should they undergo healing, the reverse process takes place. The condyloma loses its coating, becomes dry and red again, and finally disappears completely, leaving for but a short time brownish areas upon the skin.

Microscopic examination of broad condylomata discloses alterations similar to those present in the hard chancre, namely, round-cell infiltration of the cutis, and endarteritis and thickening of the walls of the blood-vessels.

Syphilitic lichen also is a papular syphilid consisting of groups of brownish-red, usually flat papules that generally appear especially upon the upper portion of the posterior aspect of the chest.

Syphilitic psoriasis is a frequent squamous syphilid. It consists in roundish, reddish-brown areas upon the skin, generally not more than one centimeter in diameter, which are covered with thin, grayish epidermic scales. They often cover the body in innumerable amount. In contradistinction from non-syphilitic psoriasis, the collections of scales are always thin, and, besides, patches of psoriasis occur not rarely upon the palms of the hands and the soles of the feet, parts that are almost unexceptionally exempt from non-syphilitic psoriasis. Actual formation of scales frequently fails to take place upon the palms of the hands and the soles of the feet. In these situations circumscribed, roundish discoloration and thickening of the epidermis are rather observed, which at times are exfoliated, leaving a circular loss of tissue.

Among the vesicular syphilids *syphilitic varicella* and *syphilitic pemphigus* may be mentioned, the former consisting of small vesicles, and suggesting the appearance of chicken-pox, while the latter is attended with the presence of large vesicles. The pustular syphilids include *syphilitic acne*, *syphilitic impetigo*, and *syphilitic ecthyma*. Impetigo and ecthyma are frequently attended with the formation of crusts upon the skin and upon the scalp when the pustules undergo desiccation.

Syphilids have certain seats of preference. Among these are the junction between the forehead and the scalp, where they are often collected in large number, and have been designated the *venereal crown*, and, also, the palms of the hands and the soles of the feet. They frequently disappear without leaving any trace of their former presence. In other instances, however, they are replaced at first by brownish, but subsequently by remarkably white areas, upon the skin, which are important in the diagnosis of antecedent syphilis. Such alterations are encountered with especial frequency upon the posterior aspect of the neck, and they are accordingly designated *syphilitic vitiligo or leukoderma* or *syphilitic nigrities or melanoderma*.

The skin is generally characterized by dryness and brittleness. Often the hair of the head falls out abundantly—**syphilitic alopecia**

—because the nutrition of the hairs is interfered with, although the hair grows again after recovery has taken place. The nutrition of the nails also suffers, and they readily become cracked and broken. Occasionally, however, the nails become diseased from the development of broad condylomata in the fold of the nail or in the bed of the nail, so that the growth of the nail is interfered with—*syphilitic paronychia and onychia.*

Of the **mucous membranes**, that of the *pharynx* suffers most frequently, becoming the seat of punctate or diffuse redness, associated with swelling and increased secretion—*syphilitic angina and pharyngitis.* Not rarely *broad condylomata* develop—that is, flat elevations of the mucous membrane, which are covered with a whitish or grayish deposit that is markedly infective. Should condylomata of the mucous membrane undergo disintegration, more or less deep loss of structure will result. Broad condylomata are encountered with especial frequency upon the tonsils and the palatine arches, but, also, upon the uvula. The patients, under such circumstances, often complain of pain in the pharynx in speaking and in swallowing; occasionally the pain extends to the ears.

Broad condylomata occur also upon the *mucous membrane of the mouth,* particularly in situations where the teeth come in contact with the mucous membrane of the cheeks, the lips, or the margins of the tongue. Inflammatory processes and broad condylomata also have been observed upon the *mucous membrane of the nose, the larynx, and the trachea.* Syphilitic patients suffer frequently from obstinate hoarseness, so that *syphilitic hoarseness of voice* is familiar even to the laity.

If the course of syphilis is left to itself, it is the rule for the syphilids to continue throughout a period of about six months, and to succeed one another in indefinite order. There then frequently follows a *tertiary or gummatous stage of syphilis.* This not rarely begins with a **gummatous iritis.** The patients often complain quite suddenly of pain and photophobia in one eye, or, occasionally, even in both, and upon the iris there will be found one or several nodules, generally of brownish color—gummata of the iris—which are usually seated close to the pupillary border. Gradually gummata may develop also in other organs. The internal viscera especially are thus affected, so that the tertiary stage of syphilis has been designated also the *stage of visceral syphilis.* This is not rarely protracted over several years, occasionally throughout the whole of life, and often terminates in the *stage of syphilitic marasmus.* In the latter *amyloid degeneration of the viscera* often takes place. If systematic treatment of syphilis is instituted from the outset, tertiary syphilis and syphilitic marasmus can generally be averted.

Rarely syphilis is confined to the hard chancre, and all other alterations fail to occur. Occasionally the symptoms of the primary and secondary stages of syphilis are so inconsiderable as to escape the observation of the patient, so that he does not come under treatment until symptoms of tertiary syphilis have developed

Syphilis has a *great tendency to recurrence,* and relapses are quite frequent, especially during the first three years after infection Under such circumstances it is impossible, even with some degree of probability, to predict whether recurrence is to be anticipated or not. Relapses generally occur the more readily the more superficial and the briefer the initial treatment, and the less frequently antisyphilitic treatment is repeated. The syphilitic patient should therefore, take especial care of himself throughout life, or, better continue under medical observation. Occasionally relapses occur even after twenty and thirty years.

Diagnosis.—The *diagnosis of hard chancre* is generally easy if **indolent enlargement of the lymphatic glands** is additionally present. In contradistinction from chancroid, the hard chancre is characterized by the fact that generally it is not multiple, that it appears as a sharply circumscribed nodule of cartilaginous hardness that neither bleeds nor is painful on pressure, and much more rarely undergoes ulceration. With regard to the differentiation between concealed chancre and *gonorrhea* reference may be made to p. 513.

Syphilids generally are characterized, in contradistinction from non-syphilitic cutaneous lesions, by their brownish-red, coppery color. They are almost never attended with itching, are frequently situated at the junction of the forehead and the scalp, on the palms of the hands and the soles of the feet, and often exhibit pleomorphism; that is, macules, papules, vesicles, and pustules frequently occur in large number side by side. The arrangement, distribution, and color of syphilids are often so distinctive that the trained eye can, to a certain extent, make the diagnosis with certainty from the general appearances. **Broad condylomata** upon the external integument and, especially, upon the mucous membrane of the pharyngeal structures and the mouth, are important in the diagnosis of syphilis.

To establish the diagnosis of antecedent syphilis the genitalia the junction of the forehead and the scalp, and the nucha should be examined for cicatrices, pigmentation, and whitish spots. Cicatrices upon the tonsils and the palatine arch are also of importance In doubtful cases the diagnosis may be based upon the results of treatment. Lesions of the skin and the mucous membranes that disappear rapidly under treatment with mercury or iodids are in high degree probably syphilitic.

Prognosis.—The prognosis of syphilis is favorable in so far as the symptoms can almost certainly be controlled by means of mercury or iodin, and there is generally no danger to life during the primary and secondary stages of the disease, with which, in the foregoing, we have dealt in detail. Syphilis but rarely terminates fatally—*malignant syphilis*. Under such circumstances there occur progressive exhaustion, extensive cutaneous lesions, and high fever, or symptoms of so-called blood-dissolution become manifest—hemorrhages into the skin, the mucous membranes, and the viscera; and this has also been designated *hemorrhagic syphilis*. It has been thought that syphilis pursues an especially severe course when it is acquired as a result of sexual intercourse with foreign races, as, for instance, by sailors in China.

The favorable prognosis of syphilis is materially modified by the fact that *relapses* occur frequently, and that it is not possible to prevent these with any degree of certainty. Nevertheless, the prognosis is much more favorable if the disease, upon its earliest appearance, is treated as soon and as thoroughly as possible. Should physicians be infected in the pursuit of their profession, they are not rarely attacked by severe syphilis, probably in part because the disease frequently is only recognized late on account of its unusual situation. The prognosis of *tertiary or visceral syphilis* is far more grave, and the condition is not rarely the cause of permanent invalidism or early death.

Treatment.—The *prophylaxis of syphilis* agrees in its fundamental principles with that of gonorrhea and chancroid (p. 436). Syphilitics should not marry within the first three years of infection, as they readily suffer relapses, and during these periods they may infect their wives and children. Syphilis is one of those infectious diseases in the treatment of which **specific remedies** can be employed, and in the primary and secondary stages *mercury*, and in the tertiary stage *iodin*, is administered for this purpose, although the use of the one does not wholly exclude that of the other.

Bloodserum-therapy has not proved successful as applied to syphilis. The blood either from syphilitic individuals or from animals (horse, dog) may be employed for the injections.

In the *primary stage of syphilis* the hard chancre should be covered once daily with *mercurial plaster*, and this treatment should be continued for weeks until the lesion is completely softened and has disappeared:

℞ Mercurial plaster, 10.0 (2½ drams).
Dose: A small bit smeared upon leather or linen to be applied.

It is advisable also to apply the plaster to adjacent enlarged lymphatic glands, or to rub these daily with *mercurial ointment*.

Attempts have been made to prevent the appearance of further syphilitic manifestations by *excision of the hard chancre*. Frequently the cicatrix left by the incision has undergone renewed induration; in any event, secondary syphilitic lesions have almost always appeared, because, obviously,

the syphilitic poison had already been distributed throughout the body prior to the operation. *Surgical removal of the enlarged lymphatic glands adjacent to the hard chancre* likewise has in no way influenced the course of the disease.

There has been much discussion whether general treatment—so-called **preventive treatment**—should be instituted during the primary stage of syphilis, and such a course has been advised against, because the secondary symptoms cannot thus be averted, and the disease is as a result only protracted for a considerable time. I have not been able to convince myself of the accuracy of this contention, and in the cases under my care have observed only benefit from early general treatment. Among all of the anti-syphilitic methods of treatment I consider the **inunction-treatment** as the most reliable and the best:

℞ Mercurial ointment, 5.0 (75 grains).
Make 30 such packets.
Dose: 1 packet daily by inunction.

The *inunction-treatment* is best carried out by having the patient take a bath daily, and subsequently rubbing the ointment in systematic order into the skin of one member, as, for instance, the leg, the thigh, the fore-arm, the upper arm, the chest, the abdomen successively. Hairy portions of the body should, so far as possible, be avoided, in order to avert inflammation of the sebaceous glands—mercurial eczema. Should this, however, occur, the inunctions in the affected situation should be omitted until recovery has taken place. Should the patient have no facilities for bathing, sponging of the skin with water and soap will have to suffice, and this should be applied both to the part anointed the day previously as well as to that to be anointed. It is important that the inunctions be continued with uniform pressure for from ten to fifteen minutes until the anointed skin has become entirely dry, in order that as much as possible of the mercurial ointment may enter the hair-follicles and be absorbed. It is true that some believe the absorption of the mercury takes place not through the skin, but through inhalation. Individuals subjected to treatment with mercurial inunctions must provide for cleanliness of the mouth in order to prevent inflammation—mercurial stomatitis. It is, therefore, necessary to rinse the mouth and pharynx with solutions of potassium chlorate after each meal:

℞ Solution of potassium chlorate, 10.0 : 200 (2½ drams: 6½ fluidounces).
Dose: To be used after each meal for rinsing the mouth and for gargling.

Systematic brushing of the teeth also should be practised, and for this purpose the following dentifrice may be employed:

℞ Fine powdered cuttle-fish bone, 50.0 (1½ ounces);
Magnesium carbonate,
Medicated soap, each, 10.0 (2½ drams).
Oil of peppermint, 5 drops.—M.
To be used as a tooth-powder.

The occurrence of mercurial stomatitis is favored by excessive smoking. It is best to interdict smoking wholly during a course of inunction-treatment. At any rate, the patient should be permitted to smoke but little, and he should use a cigar-tube.

Some patients exhibit a marked susceptibility to *mercurial intoxication* —*mercurialism*—which generally manifests itself first by a metallic taste, then by increased discharge of saliva, swelling and sponginess of the gums, and, finally, by necrotic inflammation, ulcerative stomatitis—stomacace. In neglected cases fatal general septicemia may be superadded. Occasionally adhesions form between the mucous membrane of the cheeks and the gums, which can be separated only with difficulty by operative means. Among the less common symptoms of mercurial intoxication are albuminuria, roseolous and erythematous eruptions, and neuritis. On the appearance of the first signs of mercurial poisoning the inunctions should be suspended, while the rinsing of the mouth with potassium chlorate every two hours should be continued. In addition, potassium iodid should be administered internally:

℞ Solution of potassium iodid, 10.0 : 200 (2½ drams : 6¾ fluidounces).
Dose: 15 c.c. (1 tablespoonful) thrice daily.

After the symptoms of intoxication have disappeared the resumption of mercurial inunctions may be permitted. Attempts have been made to introduce mercury into the body by other means, but it is generally recognized that the old inunction-treatment is the most reliable. The *internal administration of mercurial preparations*, which is customary especially in France, is less to be recommended in the treatment of recent syphilis than in that of relapses, particularly if these are confined to the oropharyngeal mucous membrane. I employ especially yellow mercurous iodid, and but seldom mercuric chlorid on account of its irritating properties:

℞ Yellow mercurous iodid. 0.5 (7½ grains);
Extract of licorice, sufficient to make 30 pills.—M.
Dose: 1 pill thrice daily, after meals.

℞ Mercuric chlorid. 0.1 (1½ grains);
Extract of licorice, sufficient to make 30 pills.—M.
Dose: 1 pill thrice daily, after meals.

Attempts to introduce mercury into the rectum in the form of *suppositories* were soon given up, because the absorption of mercury through the intestinal mucous membrane is far too slight. *Baths with mercuric chlorid* (10.0—2½ drams—for a full bath) are unattended with noteworthy absorption of mercury, and are of advantage only to supplement the inunction-treatment when syphilids are abundantly present.

Sublimation of calomel is scarcely practised any longer. For this purpose the patient is placed upon a chair, beneath which a spirit-lamp is lighted that heats a tin plate upon which calomel has been strewn. The patient is then covered with a woollen blanket. The calomel is vaporized when heated, and is then precipitated from the air as a fine powder upon the skin of the patient.

The *injection of mercurials* is much practised at the present day. This method possesses the advantage that the injection needs to be repeated only at intervals of several days, and that the patient while under treatment may pursue his occupation; nevertheless, abscesses and painful indurations readily develop, and dangerous, occasionally even fatal, intoxication has been observed. Soluble mercurial salts have been injected beneath the skin *subcutaneously*; insoluble preparations, on the other hand, by means of a long hollow needle into the muscles—*intramuscularly*—particularly into the gluteal muscles. Mercuric chlorid especially has been employed for subcutaneous injection, and sodium chlorid has been added to mitigate its irritating properties:

℞ Mercuric chlorid, 0.1 (1½ grains);
Sodium chlorid, 1.0 (15 ");
Distilled water, 10.0 (2½ fluidrams).—M.
Dose: 15 minims subcutaneously.

The lateral aspect of the trunk should be selected as the site for injection, as, otherwise, suppuration, and even gangrene of the skin, may readily occur. *Calomel* or *mercury in oil* has been recommended for intramuscular injection. Pulmonary emboli have been observed in a number of instances after such injections. *Intravenous infusion of mercuric chlorid* (1 : 1000) may be mentioned also as a method of treating syphilis.

The *treatment of syphilis in the secondary stage* is the same as that which has been described in the foregoing. Local treatment will, in addition, be indicated for broad condylomata. With this object in view, these formations should be painted with a solution of sodium chlorid (2.0 : 100), and by means of a camel's-hair brush they should then be covered with a thin layer of calomel. Over this absorbent cotton is placed to prevent chafing and auto-infection of opposed surfaces of the skin. During the first three years after infection it is advisable to repeat the inunction-treatment every six months, even if no relapse has occurred. Even at a later period the inunction-treatment should be repeated from every nine to every twelve months for five or six years successively. Under all circumstances the inunction-treatment should be instituted as soon as a relapse occurs.

In the *tertiary period of syphilis* iodids rather than mercurials are employed, although the latter are not contraindicated, but, on the contrary, external syphilitic ulceration generally heals with remarkable rapidity under mercurial plaster; and also mercurial inunctions frequently yield good results. Among iodids I prefer the potassium-salt, to which I like to add sodium bromid, because the former is then generally better borne. If the patient be anemic, the potassium iodid should be combined with iron:

℞ Solution of sodium bromid, 10.0 : 200 (2½ drams : 6½ fluidounces);
Potassium iodid, 10.0 (2½ drams).—M.
Dose: 15 c.c. (1 tablespoonful) thrice daily.

℞ Iron lactate,
Potassium iodid, each, 10.0 (2½ drams);
Powdered althea-root, sufficient to make 100 pills.—M.
Dose: 2 pills thrice daily.

The treatment of syphilis by means of *denutrition-cures* is no longer practised. Now and then vegetable decoctions (sarsaparilla-root, sassafras, and guaiac) are prescribed. A well-known formula is Zittmann's decoction, *compound decoction of sarsaparilla*, of which a strong and a weak form are known. In the morning before arising 500 c.c. of the stronger decoction are given, and in the evening 500 c.c. of the weaker. The principal effect of these decoctions is due to the mercury they contain.

Not rarely courses of baths are prescribed in the treatment of syphilis, particularly *sulphurous*, *saline*, and *iodin baths*. The principal value of the baths is dependent upon the inunction-treatment that is generally practised at the same time. Among sulphur-baths Aachen, Baden near Vienna, Baden and Schinznach in Aargau; and among iodin-baths Krankenheil, Tölz, Adelheidsquelle, and Sulzbrunn may be mentioned.

TERTIARY SYPHILIS.

The formation of *gummata* is distinctive of tertiary syphilis. At times these appear in the form of circumscribed tumors—*gummatous nodules*—and at other times in that of rather diffuse *gummatous infiltration*. Histologically these formations consist of round-cells enclosed within an adenoid reticulum. In recent cases they present a grayish-red appearance and an elastic consistency suggestive of that of rubber, whence the name *gumma*. These syphilitic new-growths have a great tendency to undergo caseation and suppurative breaking down, in consequence of which extensive, and often also dangerous, destruction of certain viscera may result. In contradistinction from the lesions of the secondary stage of syphilis, those of the tertiary stage occur also in internal viscera, so that this period has been designated also *gummatous* or *visceral syphilis*.

In the **treatment** iodids especially are employed, which are either administered internally, or, in the presence of external ulceration, particularly involving the mucous membranes, are also applied locally:

℞ Solution of potassium iodid,	10.0 : 200 (2½ drams : 6½ fluidounces);
Sodium bromid,	10.0 (2½ drams).—M.

Dose: 15 c.c. (1 tablespoonful) thrice daily.

℞ Potassium iodid,	1.0 (15 grains);
Pure iodin,	0.1 (1½ ");
Glycerin,	10.0 (2½ fluidrams).—M.

Apply daily with a brush.

The *administration of mercurials* is, however, not contraindicated in the presence of tertiary syphilis. On the contrary, courses of inunction with mercurial ointment, and in the presence of ulceration of the skin mercurial plaster and mercuric-chlorid baths, are indicated.

TERTIARY SYPHILIS OF THE SKIN, THE MUSCLES, THE FASCIAE, THE BURSAE, THE BONES, AND THE JOINTS.

Gummata of the skin form roundish *tumors* of soft, yet firm, occasionally almost fluctuating consistency. Should absorption take place, *depressions* in the skin not rarely remain. Frequently gummata undergo softening and disintegration. The most superficial layers of pus then often dry into a brownish-green or grayish-green crust, beneath which a second layer and then a third crust, etc., form, so that finally a collection of superimposed crusts results, which has appropriately been compared with the appearance of oyster-shells or cows' dung, and has been designated *syphilitic rupia.* If such a crust be removed from the skin, an ulcer with steep margins and a smeary, bacon-like coating will come into view. Such *cutaneous ulceration* occurs with especial frequency in places where the skin lies directly upon the bone; thus, upon the scalp, upon the forearms and the legs. Many *leg-ulcers* are of syphilitic origin. Often adjacent cutaneous ulcers become confluent, and acquire the shape of a kidney, which is distinctive of their syphilitic origin. Should cicatrization take place, the scar at first appears reddish, subsequently intensely white, with a brownish discoloration at the periphery. The renal shape of the cicatrices is indicative of their syphilitic origin.

Gummata in the muscles are uncommon, occurring with relative frequency in the *biceps of the arm.* A circumscribed, scarcely painful *tumor* can be felt, generally giving rise to shortening and contracture of the muscle. *Suppuration of a muscular gumma,* with cicatrization and permanent shortening of the muscle, may also take place; or the gumma may be absorbed and be replaced by a connective-tissue cicatrix, with the secondary changes already mentioned; or, eventually, *ossification* may take place in the gumma, and this, naturally, is likewise attended with derangement of functional activity.

In addition to gummatous nodules, *diffuse gummatous infiltration of the muscular tissue* may take place—*diffuse interstitial gummatous myositis*— which readily gives rise to compression-atrophy of muscular fibers and extensive connective-tissue hyperplasia in the muscular tissue.

Gummata of the bursæ and the fasciæ are attended variously with circumscribed globular tumors or diffuse infiltrates and thickening, and occasionally undergo suppuration.

Tertiary syphilis of the bones is attended with the presence of gummatous nodules either in the periosteum or in the bone-marrow. In either event the syphilitic tumors frequently send numerous root-like processes along the vascular channels in the structure of the bone, so that this becomes involved in the morbid process.

Periosteal gummata occur most frequently in the bones of the skull, the clavicles, the sternum, the ribs, the spine of the scapula, the bones of the forearm, and the crest of the tibia. In these situations they form firm, yet soft, elevations that often are visible also beneath the skin, and are not rarely present in groups of several close together. Should they disappear depressions upon the surface of the bone occasionally remain in their place. Sometimes they undergo ossification, so that hard projections from the bone remain permanently. Frequently, however, purulent destruction takes place, in consequence of which the structure of the bone itself is destroyed—*syphilitic caries.* In consequence of this deformities and profound disturbances occur in the structures in immediate proximity to the diseased bones. If the lesions involve the bones of the skull, for instance, the entire thickness of a portion of the calvarium is at times lost, so that the dura is exposed and its pulsation can be seen. Similar alterations occur occasionally when gummata arise from the bone-marrow. At times syphilis is followed by marked fragility of the bones—*syphilitic osteopsathyrosis.*

Little of a definite nature is known with regard to **tertiary syphilis of the joints.** *Gummatous nodules* have been observed in the joints, which occasionally undergo purulent destruction. At times articular syphilis presents the clinical picture of *deforming arthritis.* Sometimes the joints become involved secondarily after preceding syphilis of the bones. *Arthralgia* and *hydrarthrosis* also are occasionally dependent upon syphilis.

TERTIARY SYPHILIS OF THE RESPIRATORY ORGANS.

Tertiary syphilis of the nose is unfortunately not a rare disorder. Occasionally *gummata* develop upon the *alæ of the nose,* and, if they undergo disintegration, give rise to destruction of the involved structures, which is followed by marked deformity, and can be relieved only by operative measures. This condition is distinguished from *lupus* and *carcinoma of the nose* by the fact that it occurs in a syphilitic patient, and that the administration of iodin and mercury is generally followed quickly by cicatrization. *Gummatous nodules upon the nasal mucous membrane* readily give rise, with the occurrence of breaking down, to offensive nasal discharge—*ozena.* The ulcerative process may extend also to the cartilage or the bones of the nose. Occasionally *gummata* have developed upon the *cartilaginous septum of the nose.* Should they break down, a perforation is sometimes made in the septum, which may persist permanently, and is indicative of its syphilitic origin. *Gummata of the cartilaginous portion of the bridge of the nose* are followed, after previous breaking down, by sinking in of the anterior portion of the nose, as a result of which the nose becomes increased in width, and its lowermost portion and the two nasal orifices are thrown into a single wide fissure.

Gummata of the nasal bones further are of serious significance. If the bony roof of the nose is involved, and purulent softening takes place, the destroyed and loosened portions of bone can occasionally be felt to rub against one another and to crepitate. Often the bony roof of the nose falls in, so that the upper portion of the nose exhibits a deep depression. The designation "*sheep's nose*" or "*saddle-nose*" has, therefore, also been employed, and from which the experienced eye can, at a glance, determine the existence of syphilis. Occasionally the *turbinates* and the *ethmoid bone* are the seat of gummata. Should these undergo purulent destruction, the patient frequently suffers from a *putrid nasal discharge*—*syphilitic ozena*—whose dependence upon bone-destruction will be rendered certain if the patient expels particles of bone (occasionally of considerable size) in blowing the nose, and if, by means of a sound, rough and detached bone can be demonstrated. Disease of the ethmoid bone is attended further with the danger that the olfactory nerve, and with it the sense of smell, may be destroyed—*syphilitic anosmia;* or that the cerebral meninges and the brain itself may be involved.

Tertiary syphilis of the larynx occasionally develops insidiously and without symptoms, so that, for instance, the *epiglottis* may be found accidentally on laryngoscopic examination transformed by gummata into a cylindric body almost as thick as a thumb. In other cases, however, dangerous and painful symptoms appear. The patient may be hoarse, the expectoration purulent, at times extremely offensive in odor, and speaking and swallowing are attended with intense pain. These manifestations appear when gummata of the larynx undergo purulent softening, and the true vocal bands are involved in the morbid process. The destructive process extends not rarely to the perichondrium, and gives rise to *laryngeal perichondritis* and its attendant dangers. Occasionally *acute edema of the glottis* sets in suddenly, with the danger of suffocation. Should cicatrization take place in broken-down gummata, *changes in the voice* frequently remain permanently because the free margins of the vocal bands are the seat of contracted scars. At times *adhesions* take place between the *vocal bands*, so that only a small, frequently too narrow, orifice remains for the current of air, and the enlargement of which gradually by means of bougies should be attempted. Should this fail, or should danger of suffocation threaten, resort will have to be had to intubation of the larynx or to tracheotomy. Some patients are condemned to wear a tracheal cannula throughout the remainder of life.

Syphilis of the larynx may readily be confounded with *tuberculosis of the larynx*, although the former is unattended with the presence of tubercle-bacilli in the sputum. Occasionally tuberculosis is associated with syphilis of the larynx. Confusion with *carcinoma of the larynx* and with *leprosy* is possible also, but carcinoma is not cured by the administration of potassium iodid and mercury, and leprosy is attended with distinctive lesions of the skin.

In addition to internal *treatment*, local treatment also should be employed. Insufflations of calomel or of iodoform, applications of iodin and potassium-iodid glycerin, and inhalations of potassium iodid and mercuric chlorid have been practised.

Tertiary syphilis of the trachea and the bronchi is frequently manifested by *purulent catarrh* if gummatous nodules of the mucous membrane break down. Under such circumstances the lung may be secondarily involved from the entrance of purulent secretion into the pulmonary alveoli. Should gummata undergo cicatrization, symptoms of *tracheal* or *bronchial stenosis* occasionally appear. Disorders of the latter variety develop also in some cases from the fact that rings of cartilage become involved in the destructive process, and the trachea or the bronchi, as a result, become constricted.

Tertiary syphilis of the lungs at times wholly resembles chronic pulmonary tuberculosis by reason of its insidious, almost febrile onset, sweats, progressive pallor and emaciation, hemoptysis, signs of infiltration in the upper portions of the lungs; but tubercle-bacilli are not found in the sputum in cases of syphilis of the lungs, and, besides, the manifestations subside completely and with remarkable rapidity after the administration of mercurials and iodids. Occasionally tertiary syphilis of the lungs is attended with chronic intra-alveolar or interstitial pneumonia, although with regard to this little of a reliable nature is known.

Tertiary syphilis of the pleura occasionally gives rise to thickening of the pleura in places, which undergoes cicatricial contraction, and leads to the development of *deforming syphilitic pleuritis.*

Occasionally diffuse infiltrates or gummatous nodules develop in the *mammary gland* not only in women, but also in men, and which yield to the usual antisyphilitic treatment.

TERTIARY SYPHILIS OF THE DIGESTIVE ORGANS.

Gummata of the tongue may form tumors up to several centimeters in length, generally painless on pressure, and giving rise also to enlargement and disturbances in the functional activity of this organ. Should they undergo purulent softening, the tongue may be destroyed. Gummata of the tongue may be readily confounded with *solitary tubercles* or *carcinoma of the tongue,* although in contradistinction from tuberculosis tubercle-bacilli cannot be demonstrated in the purulent discharge, and in contradistinction from carcinoma it should be emphasized that this condition occurs principally in elderly persons, that pain is generally present, and that the adjacent submaxillary lymphatic glands are likely to be involved in carcinomatous degeneration, and therefore can be felt to be indurated and enlarged. Gummata of the tongue also are usually single, and disappear under the use of iodids and mercurials.

Tertiary syphilis of the palate and the pharynx involves at times the soft structures, and at other times the bony parts. *Gummata of the hard palate* can be felt through the mouth as soft but firm enlargements. Should purulent destruction take place, the gummata rupture occasionally into the nose and the mouth at the same time, and a perforation in the hard palate results, through which a probe can be passed from one cavity into the other. In eating and drinking food then readily enters the nasal cavity, inducing sneezing and blowing of the nose. In addition, speech is affected, and acquires a nasal character. Many patients overcome the resulting difficulties by making a plate of bread-crumbs, with which they keep the artificial opening closed for the day. The same purpose may be accomplished by obturators. The obliteration of the opening by surgical measures is not always easy.

Gummata of the soft palate and of the *mucous membrane of the pharynx* often give rise to most extensive destruction by reason of purulent softening. Should cicatrization subsequently take place, the palatine arches occasionally become adherent to the posterior wall of the pharynx, forming a sort of diaphragm between the pharynx and the nasal cavity, and at times so reducing the orifice of communication between these two spaces that speech, respiration, and ingestion of food are greatly interfered with. Such deformities can be corrected only with difficulty by operation.

Gummata of the esophagus are rare. Occasionally the cicatrices resulting from them give rise to stenosis of the esophagus.

Gummata of the gastric mucous membrane are without marked clinical significance, and give rise variously to the symptoms of gastric ulcer or of a gastric cicatrix.

Gummata of the intestinal mucous membrane develop with especial frequency upon the mucosa of the rectum. They often give rise to symptoms of *purulent proctitis—suppurative inflammation of the rectum*—which is characterized by rectal tenesmus, and purulent, even bloody, thin stools, and which may readily be confounded with dysentery. Should gummata of the rectum subsequently undergo cicatrization, *stenosis of the rectum* not rarely persists.

Tertiary syphilis of the liver is at times attended with the development of *circumscribed gummata*, so that the surface of the organ is often the seat of numerous round projections, which may readily be confounded with carcinoma of the liver. The nodules are not tender on pressure. Jaundice is wanting, or is generally present in but slight degree. In doubtful cases in which the question of carcinoma or gumma of the liver arises iodids and mercurials should be prescribed, and a decision given in favor of syphilis of the liver if the tumors diminish considerably in size or even disappear entirely. Occasionally gummata of the liver undergo suppuration, caseation, or calcification. In some cases tertiary syphilis of the liver is manifested by *diffuse infiltration* of the organ, in consequence of which the liver-cells in considerable portions of the viscus are entirely lost. Not rarely gummata and connective-tissue hyperplasia are associated. Some portions of the liver become constricted off, and the organ acquires a multilobulated appearance—*syphilitic lobulated liver.* The relations between syphilis and *cirrhosis of the liver* have already been discussed in Vol. I., p. 334. Occasionally syphilis has constituted the basis for *chronic perihepatitis*, resulting in numerous adhesions with adjacent structures.

Tertiary syphilis of the pancreas is without clinical significance, and is characterized either by the formation of circumscribed gummata or by diffuse connective-tissue hyperplasia.

Reference to *tertiary syphilis of the spleen* may yet be appended. This condition is attended with the formation of circumscribed nodules, causing irregularity of the surface of the spleen, or with diffuse connective-tissue proliferation, or with perisplenitic alterations.

TERTIARY SYPHILIS OF THE GENITO-URINARY ORGANS.

Tertiary syphilis of the testicle is the most frequent and the most important syphilitic lesion of the genito-urinary apparatus. The testicle is at times the seat of gummata. The diseased organ becomes increased in size, and infiltrated with roundish, indurated nodules, which are but slightly sensitive on pressure. The condition has been designated also *syphilitic sarcocele.* Suppuration rarely takes place in gummata of the testicle. Should the new-formation disappear under antisyphilitic treatment, remnants of indurated connective tissue generally persist. It is noteworthy that men, even with bilateral sarcocele, are capable of procreation, obviously because only small portions of healthy testicular tissue are necessary for the purpose. The condition is occasionally mistaken for *carcinoma of the testicle*, although this affection is generally painful, and the adjacent inguinal lymphatic glands are usually involved in carcinomatous degeneration, and are enlarged and indurated; and, finally, iodids and mercurials are without effect, while they cause the disappearance of gummata of the testicles. Confusion with *tuberculosis* also is possible. It should be noted especially that tuberculosis is situated in the epididymis, while the syphilitic lesions generally avoid this organ. Besides, the therapeutic influence of iodin and mercury must be taken into consideration. In addition to the formation of gummata, tertiary syphilis of the testicles is occasionally manifested by *diffuse connective-tissue hyperplasia.* Further, both processes are not rarely associated; although gummata develop in other portions of the genito-urinary apparatus, they are only of subordinate significance.

TERTIARY SYPHILIS OF THE CIRCULATORY ORGANS.

Gummata and diffuse connective-tissue hyperplasia have been encountered in the **myocardium** in cases of tertiary syphilis, and they have occasionally led to a fatal termination amid symptoms of progressive weakness of the heart muscle

Gummata have been observed also in the **pericardium** in exceptional instances, and at times they have been enclosed within pericardial adhesions.

A few instances are on record of gummata upon the intima of the **arteries and veins.** Under such circumstances thrombosis and gangrene of an extremity may occasionally occur.

Syphilis is properly considered a cause for *endarteritic alterations*, and has been held responsible for the formation of *aneurysms.*

TERTIARY SYPHILIS OF THE NERVOUS SYSTEM.

The **peripheral nerves** are but rarely the seat of syphilitic lesions, although they are often involved as a result of pressure from contiguous structures (bones, meninges). Muscular spasm, paralysis, neuralgia, and anesthesia may then appear, accordingly as the motor or the sensory nerves are involved in irritation or in paralysis. The nerves of special sense also are, under some conditions, involved.

Syphilis of the spinal cord is by some clinicians considered not at all a rare disease, but it must be admitted that convincing anatomic findings are known in but a small number of instances. It would appear as if the cases were dependent with especial frequency upon alterations in the vessels of the spinal cord. In the first place, *syphilitic spinal meningitis* and *meningomyelitis* should be considered. These occur in an acute, but more frequently in a chronic, form, and in their symptomatology are scarcely distinguishable from the same affections dependent upon non-syphilitic causes. Anatomically, it has been pointed out that marked thickening and adhesion of the spinal meninges are generally prominent. In some cases of chronic myelitis muscular contractures predominate, and the tendon-reflexes are so exaggerated that the clinical picture resembles that of *spastic spinal paralysis.* Further, it should be emphasized that some cases of myelitis are probably less purely inflammatory than thrombotic and necrotic in origin, and they should rather be designated *softening of the spinal cord* or *myelomalacia.*

Syphilis is correctly considered the most frequent cause of *wasting of the spinal cord—tabes dorsalis*—and it also plays an important etiologic *role* in the development of *multiple cerebrospinal sclerosis. Anterior poliomyelitis* also is attributed by some clinicians to syphilis. *Gummata in the substance of the spinal cord* are of rare occurrence. Occasionally syphilis gives rise to the development of *acute ascending spinal paralysis*, thus to a spinal neurosis, in explanation of which no anatomic lesion is as yet known.

The *prognosis of syphilitic disease of the spinal cord* is by no means favorable, as antisyphilitic treatment is at best capable of preventing further progress in the alterations, but such nervous tissue as has been destroyed as a result of hemorrhage or softening is not capable of being restored by any remedy. Improvement is conceivable only when the functions of the spinal cord in the neighborhood of diseased foci are interfered with by pressure or edema, and the disturbances subside in consequence of partial absorption of the morbid products.

Attention should further be called here to the fact that occasionally the spinal cord becomes involved secondarily to *syphilitic lesions of the vertebral column* (gummata, exostoses, caries).

Cerebral syphilis is, unfortunately, quite a common disease, and its development is believed to be favored by the physical and mental strain of modern life. Individuals who engage in great cerebral activity, as, for instance, scholars, suffer also from cerebral syphilis with especial frequency. A neurotic predisposition, alcoholism, and injuries to the head may also be mentioned as contributory etiologic factors. The danger of the development of cerebral syphilis is the greater the more superficially antisyphilitic treatment has been carried out. It is true it has also been maintained under such circumstances that courses of antisyphilitic treatment too frequently repeated favor the occurrence of cerebral syphilis. Occasionally symptoms of cerebral syphilis appear as early as the end of the first year after infection. It is the rule, however, for five, ten, occasionally more than twenty, years of perfect health to have intervened until suddenly symptoms of cerebral syphilis appear.

Cerebral syphilis is attended with purely functional disturbances, or gummata develop in the meninges or in the structure of the brain, or, finally, the cerebral arteries become the seat of obliterating endarteritis, which directly, or through the formation of thrombi, gives rise to vascular occlusion, and, as a result, to necrotic softening in the related portions of the brain.

Gummata of the cerebral meninges are seated with especial frequency upon the dura mater at the base of the skull, or upon the convexity of the brain close to the falx cerebri. At times they appear in the form of circumscribed nodules, at other times in that of diffuse infiltrates. Not rarely they undergo caseation, in consequence of which there may be danger of mistaking them for caseous tubercles. The same statement is applicable to caseous gummata of the cerebral tissue. Gummata of the basal dura not rarely exert compression upon the cerebral nerves arising at the base of the brain, and, in consequence, give rise to peripheral paralysis of the cerebral nerves.

The artery of the fossa of Sylvius and the basilar artery are involved in endarteritic changes with especial frequency, so that bulbar, pontine, and aphasic disturbances are frequently encountered in cases of cerebral syphilis. Occasionally, in addition to endarteritis, gummata also have been found present in the adventitia and the media of the diseased arteries.

Among the purely *functional cerebral disturbances sleeplessness—agrypnia*—is to be considered, and of which syphilitic patients complain with exceeding frequency. Many become *neurasthenic* and *hypochondriacal* in a marked degree, believing that any lesion of the skin must be of syphilitic origin, and they run from one physician to another in the hope of securing comfort. Such alterations are at times followed by marked *psychopathic states.* The conviction is steadily growing at the present day that *progressive paralysis of the insane* is, in the vast majority of cases, dependent upon antecedent syphilis, whence also its frequent association with tabes dorsalis.

As has been mentioned, *paralysis of cerebral nerves* is frequently dependent upon gummatous changes at the base of the brain. The ocular nerves are involved with especial frequency, and their paralysis not rarely recurs from time to time, so that with good reason the diagnostic dictum has been set up that recurrent paralysis of the ocular muscles, without demonstrable extraneous cause, is indicative of a syphilitic origin. Occasionally, however, almost all of the cerebral nerves upon one side are paralyzed, and even the nerves upon the opposite side may be involved, and the gradual progress of the paralysis may be distinctly noted.

Endarteritic alterations in the cerebral arteries often occur in the artery of the fossa of Sylvius on the left side, and, therefore, frequently give rise to right-sided hemiplegia and aphasia. At times the symptoms set in suddenly—apoplectiform; at other times they develop gradually. Not rarely exacerbations and remissions occur repeatedly in alternation, obviously because the brain has temporarily adapted itself to a deficiency of blood until the vascular obstruction is again increased. Monoplegia, or aphasia alone, also occurs, however, in the course of cerebral syphilis if the circulation in circumscribed areas of the cerebral cortex only is disturbed. It is especially characteristic of cerebral syphilis for the symptoms frequently to recur and disappear. Individuals with cerebral syphilis not rarely exhibit *coma* for days and weeks, and occasionally death occurs in this condition. Others are troubled with *vertigo, headache,* or *tremor. Circumscribed gummata of the brain* give rise to symptoms of cerebral tumor, such as have been described on pp. 69–71.

The *diagnosis of cerebral syphilis* is not always easy, for no distinctive cerebral symptoms are known. It is naturally important to obtain evidence from the history, or from the presence of cicatrices and pigmentary lesions, of the certainty of infection of the patient with syphilis. A remarkably smooth appearance of the dorsum of the tongue, in consequence of atrophy of the papillæ, is correctly considered an important post-syphilitic manifestation. Occasionally examination of the eye will yield the desired information. Disseminated choroiditis, optic neuritis, and endovascular and perivascular changes in the retina especially should be looked for. In doubtful cases it is useful practically to assume a syphilitic origin and to institute antisyphilitic treatment, as this may be of much service and will scarcely ever do serious harm. The *prognosis* of cerebral syphilis corresponds with that of spinal syphilis (pp. 529 and 530). Recently attempts have been successfully made to remove localizable gummata by operative means. The principal remedies for cerebral syphilis, nevertheless, remain mercury and iodin.

HEREDITARY SYPHILIS.

Etiology.—Syphilis in the parents may be transmitted to the children if at the time of conception the father or the mother, or perhaps both, should be suffering from the symptoms of primary or secondary syphilis. If, however, tertiary syphilitic lesions exclusively are present, hereditary transmission of syphilis does not take place, although the children are, under such circumstances, often poorly developed, and are frequently attacked by tuberculosis and scrofulosis. The hereditary transmission takes place only to the succeeding generation, so that the grandchildren of syphilitic persons are exempt from hereditary syphilis. Not rarely only a portion of the children suffer from hereditary syphilis, while the others remain healthy, and this fact is to be explained upon the supposition that the conception of the first has taken place at a time when syphilitic symptoms were present, while that of the others occurred in a free interval. Occasionally, as a matter of accident, syphilitic and non-syphilitic children are born in alternation. Often the older children are syphilitic, while the younger are free from syphilis. The explanation resides in the fact that relapses of syphilis in the parents occur the less commonly the longer the interval from the time of infection.

Should hereditary syphilis, as is the rule, be derived from syphilis in the father, it may happen that the mother has not been infected in sexual intercourse, although she gives birth to a child with hereditary syphilis. It has been maintained under such circumstances that even if the mother exhibits no syphilitic manifestations, nevertheless a sort of immunity to syphilitic infection has been developed in her, so that, for instance, she may nurse her syphilitic child at the breast without danger of infection. This is the so-called *law of Colles,* to which, however, exceptions occur, and practically it is well not to place too much dependence upon it.

It occasionally happens that the parents are healthy at the time of conception, and that the mother acquires syphilis only during pregnancy. Should syphilitic infection of the mother take place within the first five months of pregnancy, the fetus may become syphilitic subsequently; at a later period, on the other hand, the fetus frequently, though not invariably, remains healthy. Naturally, however, there is danger that infection of the child by the mother may take place even after birth. Further, remarkable and irregular phenomena occur in the course of hereditary syphilis. Thus, for instance, it is known that of twins only one child may be syphilitic, while the other is entirely well. It was formerly supposed that individuals with hereditary syphilis were immune to subsequent infection with syphilis—Profeta's law—although it is certain that exceptions to this rule occur.

Symptoms and Diagnosis.—Hereditary syphilis is a frequent cause of **miscarriage**, and for this reason inquiry as to syphilis in the parents must always be made if frequent miscarriages occur. This is especially the case if the children are stillborn and macerated. On examination of the umbilicus it will frequently be found under such circumstances that the umbilical arteries are constricted by periarteritic and endarteritic proliferation, and often also are occluded by thrombi. Similar alterations are present in the umbilical vein, and explain the death of the fetus, which does not receive sufficient blood from the mother. Changes are present, also, in the placenta, especially interstitial connective-tissue hyperplasia and gummata. Frequently firm adhesions have formed between the placenta and the internal surface of the uterus. In other cases living children are born, but these exhibit **deficient development**—*syphilitic pediatrophy*. The face is pale, sunken, and aged-looking, the children cry with a feeble voice, evince no desire for food, and must be nourished by force. The skin is thrown into folds and is wrinkled, and frequently, especially upon the palms of the hands, the soles of the feet, and the buttocks, is thin and glistening as if varnished. Death often occurs within a few days after birth in consequence of progressive exhaustion.

Hereditary syphilis is occasionally attended with **peritoneal dropsy—ascites**—of such marked degree as to constitute an obstruction to labor. Should death occur, endophlebitic changes in the portal vein, and usually, also, connective-tissue hyperplasia in the liver, are encountered upon post-mortem examination. Intense **jaundice—icterus**—in the newborn is also often dependent upon syphilitic changes in the liver (gummata, connective-tissue hyperplasia).

Frequently the children are strong and healthy at birth, but syphilitic lesions appear in the course of from four to eight weeks. Occasionally these do not occur before the lapse of from four to six months, rarely later. The designation *late hereditary syphilis* has been applied to cases in which the first symptoms of hereditary syphilis have appeared between the eighth and the fifteenth year of life, in women even still later. Although some of the cases described depend upon confusion with tuberculosis, it can, on the other hand, scarcely be doubted that late hereditary syphilis does occur, it is true, as a rare disorder.

It is distinctive of hereditary syphilis that it sets in at once with secondary, often even with tertiary symptoms, and that it is unattended with a hard chancre. The first lesions are likely to consist in **syphilids** of the skin and the mucous membranes. Not rarely the entire body is covered with numerous *roseolæ*. *Syphilitic pemphigus* also is not a rare manifestation. It is characteristic of its syphilitic nature that it frequently involves the palms of the hands and the soles of the feet. The *broad condyloma* occupies a prominent place also in the symptomatology of hereditary syphilis on account of its frequency and diagnostic importance. It is encountered with especial frequency surrounding the anus, upon the scrotum and the umbilicus. Broad condylomata are frequently concealed at the angles of the mouth behind *fissures*, and it should, therefore, always be borne in mind that fissures of the corners of the mouth in the newborn are often dependent upon hereditary syphilis. The same statement is applicable to **occlusion of the nares** and to **nasal discharges** resulting from syphilitic catarrh of the nasal mucous membrane.

In some children one or more members can be moved but imperfectly, if at all—**syphilitic pseudo-paralysis.** Careful examination will frequently disclose **syphilitic bone-disease—syphilitic osteochondrosis**—as the cause. Under such circumstances inflammatory changes have taken place in the epiphyseal cartilage, leading to destruction, in consequence of which the diaphysis and the epiphysis become separated—*syphilitic detachment of the epiphysis.*

Often *tertiary syphilitic symptoms* appear exceedingly early, resembling wholly the tertiary symptoms of acquired syphilis, and often giving rise to the most serious deformities and disturbances of function in different viscera. These not rarely continue from earliest childhood to complete maturity, and are often confounded with tuberculous disease. As a matter of course, however, tubercle-bacilli are never present in the discharges from breaking-down syphilitic formations.

In the diagnosis of hereditary syphilis importance has been attached in doubtful cases to the presence of Hutchinson's **triad.** This includes, in the first place, a malformation of the upper central permanent **incisor teeth,** whose contiguous borders, and in correspondence therewith whose chewing surfaces also, converge inward, occasionally, also, diverge outward, are deficient in length, and besides present a gradually eroded chewing surface, with its convexity upward. In addition, children with hereditary syphilis frequently exhibit **parenchymatous keratitis**; and, finally, they not rarely suffer from unilateral or bilateral **deafness.**

The *duration of hereditary syphilis* occasionally extends throughout the whole of life, and *relapses* take place from time to time. Not rarely *tuberculous lesions* are superadded to the syphilis. *Amyloid degeneration* also is not rarely a complication. The children often exhibit pallor, and examination of the *blood* discloses a reduction in the number of red corpuscles and in the percentage of hemoglobin. The patients often remain backward in growth, and exhibit deficient development of the genitalia and the breasts. Hereditary syphilis often exerts an injurious effect upon the *central nervous system*, and gives rise to *idiocy*, *epilepsy*, *hydrocephalus*, *spastic spinal* and *cerebral paralysis*, and the like. *Progressive paralysis of the insane* also has been observed to occur in children with hereditary syphilis.

Prognosis.—The prognosis of hereditary syphilis is serious, as many children die in consequence of progressive exhaustion soon after birth, while others remain sickly, deformed, and permanently deficient mentally and physically. It is most important for the family physician to recognize syphilis in the parents, for by means of a thoroughly carried out course of inunction and treatment it is not rarely possible to prevent the previously repeated miscarriages and to bring about the birth of healthy children.

Anatomic Alterations.—Among the anatomic alterations of hereditary syphilis, lesions of the epiphyseal cartilages are of preëminent diagnostic significance. The diseased cartilages appear increased; their margins are not straight, but pursue an irregular course in the direction of the osseous tissue, and, besides, they are the seat of cheesy-yellow tissue involved in purulent softening. Further, gummata and diffuse connective-tissue infiltrates in various viscera should be looked for. Thickening and adhesions are frequently encountered in the serous membranes, and the serous cavities not rarely contain hemorrhagic effusions. The lungs are occasionally the seat of pneumonic alterations, presenting a light-gray or whitish color, so that the designation *white pneumonia* has been employed. Microscopic examination discloses that the condition is dependent in part upon interstitial connective-tissue hyperplasia, in part upon proliferation, exfoliation, and fatty degeneration of the alveolar epithelial cells. The liver exhibits gummatous and interstitial connective-tissue hyperplasia with especial frequency. Often chronic *perihepatitis* has developed, and [illegible] given rise to numerous and often firm adhesions between [illegible] serous coat of the liver and adjacent structures, particularly [illegible] diaphragm. Occasionally the pancreas is indurated in [illegible] of interstitial connective-tissue hyperplasia. Similar [illegible] are frequently appreciable in the lymph-follicles of the [illegible] mucous membrane. Gummata occur in the kidneys [illegible] of the adrenal bodies, and in the latter situation [illegible] frequently undergo caseation. It was formerly [illegible] cheesy foci in the thymus gland were [illegible] syphilis, although under such circumstances confusion with normal processes, particularly with accumulation of secretion, has occurred.

Treatment.—Hereditary syphilis would naturally be prevented if it were possible to eradicate acquired syphilis. In any event, syphilitics should not marry before the lapse of three years after infection, and then only if no syphilitic symptom has been present for six months. Should new syphilitic symptoms, nevertheless, again appear, a thorough course of inunction-treatment should at once be instituted, and all sexual intercourse be forbidden until a complete cure has been effected. Should syphilitic mothers give birth to healthy children the former should not nurse the latter, so as not, through the intimate association with the child, to convey syphilis subsequently to the latter, as, for instance, through broad condylomata, which often develop upon the nipples. The milk does not contain infective matter. Pregnant women suffering from syphilis should be subjected to antisyphilitic treatment in order to restore the mother, and possibly also the fetus, to health as speedily as possible. Should a healthy woman give birth to a syphilitic child, it is the duty of the physician to inform the mother that application of the child to the breast will expose her to the danger of syphilitic infection, as, for instance, from broad condylomata on the lips of the child. If artificial nourishment be provided, on the other hand, the child will be exposed to the danger of death from exhaustion. The mother will have to decide between these two possibilities, and if she possess the proper character she will assume the risk of infection. The physician should never advise the employment of a healthy wet-nurse, for she likewise will be exposed to the danger of infection. On the other hand, no objection can be raised to the employment of a syphilitic wet-nurse. Under such circumstances both the nurse and the child should receive antisyphilitic treatment.

Mercury and **iodin** are the most efficient remedies in the treatment of hereditary syphilis also. Inunction with mercurial ointment and injections of mercurial salts would not be tolerated by the skin of the newborn and the infant, so that resort must be had to internal administration and to baths. Internally calomel and black mercurous oxid especially are to be recommended :

℞ Mercurous chlorid,	0.01 (⅙ grain);
Sugar,	0.3 (4½ grains).—M.

Make 10 such pills.
Dose: 1 pill thrice daily.

℞ Mercurous oxid,	0.01 (⅙ grain);
Sugar,	0.3 (4½ grains).—M.

Make 10 such pills.
Dose: 1 pill thrice daily after meals.

For baths mercuric chlorid (3.0—45 grains—to a full bath) is employed, although care should be taken to prevent the children from swallowing any of the water or its being splashed into the eyes. Symptoms of tertiary hereditary syphilis should be treated in the same manner as similar symptoms in cases of acquired syphilis.

IV. LEPROSY.

Etiology.—Leprosy is at the present day principally a *disease of the tropics.* In the middle ages it was widely prevalent also in Europe, and only through rigid quarantine measures has it been possible to confine it to small areas of Norway, Sweden, Iceland, Russia, Roumania, Greece, Italy, etc. In recent years new foci of leprosy were found to have developed in Germany (in the vicinity of Memel) and in the south of France; and, in any event, governments will do well to keep leprous patients under careful observation in order to prevent epidemic distribution of the pestilence.

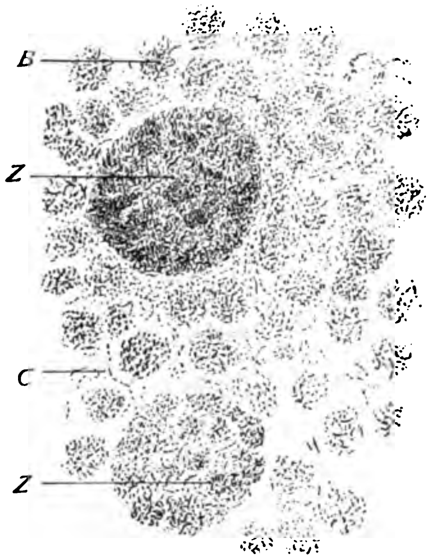


Fig. 80.—Bacilli shown in a section of the tongue from a case of tubercular leprosy; magnified 600 times. The bacilli are extracellular: *B*, bacilli in groups; *Z*, *Z*, zoöglear masses, large rounded masses of bacilli; *C*, bacilli in chains (Leloir).

Leprosy is without doubt an *infectious disease*, which has been successfully transmitted to criminals and also to healthy persons in the act of vaccination. The *exciting agent* of leprosy is the **leprosy-bacillus,** which was first demonstrated by Hansen (1880) in leprous tissues. In their reactions to stains leprosy-bacilli resemble closely tubercle-bacilli, and they often lie together in cells—leprosy-cells—in groups (Figs. 80 and 82). They are found, among other situations, in large number in scales of the skin, in the saliva, and in the nasal secretion. Possibly they gain entrance into the body by inhalation, as leprosy of the nose is one of the earliest and most constant manifestations. The disease occurs *between the twentieth and fortieth years of life,* and children are almost always exempt.

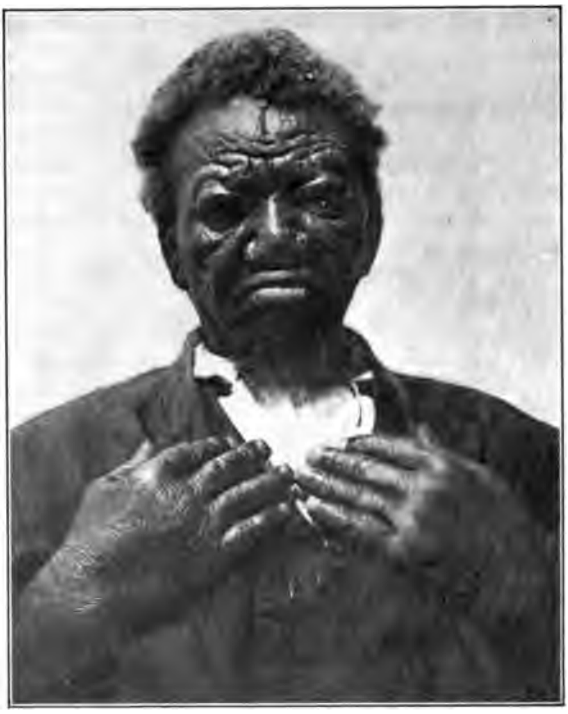


FIG. 81.—Tubercular leprosy in a negro (Dyer).

Symptoms and Prognosis.—The *period of incubation of leprosy* may apparently be several years. *Prodromes* are frequently manifested in transient febrile movement, which is often attributed to malaria. Two varieties of leprosy have been distinguished, and they have been designated *cutaneous leprosy* and ***nervous leprosy;*** although, in the further course of the disease, the one variety generally passes over into the other.

In cases of *cutaneous leprosy* the earliest alterations appear in the skin. Here and there **red spots** develop, which in part disappear, but in part are replaced by brownish discoloration. In addition, **thickening of the skin** occurs, either in a diffuse manner or in nodular distribution—*nodose*, *tuberous*, or *tuberculous leprosy*. This thickening of the skin also may subside, but sometimes it persists. In consequence deformities occur, especially in the *face*. The chin, the lips, the nose, the eyelids, and the auricles become increased in size, and acquire a rigid, mask-like appearance. The face has been compared with that of a lion or a satyr, and the designations *leprous leontiasis* and *satyriasis* have therefore been employed. Some of the leprous infiltrates and nodules begin to break down, and **leprous ulcers** thus result upon the skin, which secrete small amounts of pus, present indolent granulations, and but slowly undergo cicatrization. The **hair** generally falls out, and the absence of the eyebrows and the eyelashes is especially noteworthy. The **external lymphatic glands** gradually and progressively become enlarged.

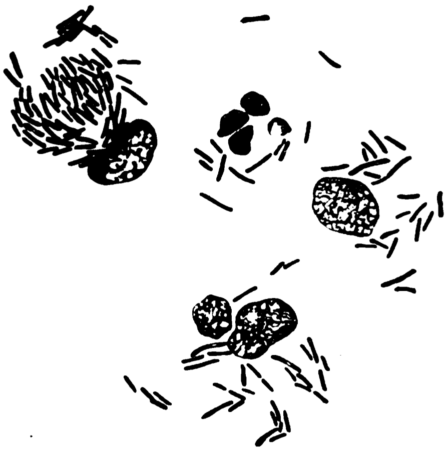


FIG. 82.—Leprosy-bacilli from nasal secretion: fuchsin-methylene-blue stain; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

Diffuse leprous infiltrates and nodules develop not only upon the external skin, but also upon the **mucous membranes**, with especial earliness and constancy upon the nasal mucous membrane, but also upon that of the pharynx, the larynx, and the deeper air-passages. In consequence stenosis of the nares and the upper air-passages frequently results. The voice becomes hoarse and finally toneless. In addition, however, *ulcers* not rarely form, and frequently they perforate the cartilaginous septum of the nose. On post-mortem examination the *internal viscera* also are generally the seat of leprous infiltrates and nodules, although these often remain concealed during life. It is noteworthy that frequently the testicles are the seat of leprous new-formations, and they undergo progressive atrophy. Leprous new-formations have been observed also upon the *conjunctiva*, the *cornea*, and the *iris*.

Nervous leprosy generally begins with circumscribed or diffuse thickening of the **peripheral nerve-trunks**, earliest and most constantly in the ulnar nerve, just above the internal condyle of the humerus, and in the great auricular nerve. Circumscribed areas of the skin then present **hyperesthesia.** Gradually this is replaced by **cutaneous anesthesia,** which frequently is at first partial and involves especially painful and thermic sensibility—*anesthetic leprosy.* Gradually **trophic** and **vasomotor disturbances** are superadded. Vesicular elevations appear upon the skin—*leprous pemphigus*—in the fluid of which leprosy-bacilli can be demonstrated. Occasionally **perforating ulcer of the foot** develops. It may also happen that the phalanges of the fingers and the toes are exfoliated after painless ulceration—so-called *mutilating leprosy.* The skin often acquires a brownish or a bright-white appearance in places—*macular leprosy.* Occasionally **edema** of the hands and the feet develops, the overlying skin being deeply cyanotic, whence the name blue edema. Such *muscular paralysis* as is present exhibits peripheral characteristics. The paralyzed muscles exhibit also increased mechanical irritability and degenerative electric reaction. Paralysis is often complicated by *muscular contractures and atrophy.*

The *course of leprosy* is chronic, and often extends over many years. Death occurs, in the presence of progressive exhaustion or of leprous ulcers, as a result either of septicemia or of intercurrent disease, as, for instance, pneumonia. Although leprous new-formations may in part undergo involution, permanent recovery, however, is extremely rare, so that the *prognosis* is unfavorable.

Diagnosis.—A positive diagnosis of leprosy is possible only by bacteriologic methods. Leprosy-bacilli can be demonstrated in the nasal mucus, the sputum, the urine, and the blood of leprous patients. The contents of cantharidal blisters also have usually been examined for leprosy-bacilli with success. In a patient under my care innumerable leprosy-bacilli were found in the scales from the skin. From the clinical manifestations alone leprosy might readily be confounded with **tuberculosis.** Nervous leprosy is not rarely attended with symptoms of **syringomyelia** and **Morvan's disease** (Vol. I., pp. 566 and 567).

Anatomic Alterations.—Diffuse infiltrates and circumscribed nodular accumulations of round cells developing in all of the viscera are distinctive of leprosy. Within these leprosy-bacilli are encountered, generally enclosed in cells, some of which have developed into polynuclear giant-cells, and are filled with vacuoles. In cases of nervous leprosy, peripheral nerve-trunks are the seat of leprous formations, with resulting degenerative destruction and loss of nerve-fibers. Leprosy-bacilli have been found also in the brain and the spinal cord.

Treatment.—In order to prevent the spread of leprosy by inoculation the most reliable *prophylactic measure* is rigid segregation of the patients in special hospitals (leproseries). In addition the sputum, the urine, the nasal secretion, and the cutaneous scales of the patient should be thoroughly disinfected. Leprosy-bacilli have been found also in the sweat.

No *specific remedy* for the treatment of leprosy is as yet known. Recently, *curative serum* has been employed, although the experiences thus far reported from its use are by no means encouraging. Among internal remedies successful results have been reported especially from the use of salicylic acid and sodium salicylate. Inunctions with salicylated ointment, or with ichthyol, and sulphurous baths also have been warmly praised. Chaulmoogra oil (in doses up to 15.0—½ fluidounce—daily) has been employed in Japan with success. Attention should be especially directed to sustaining the strength of the patient by means of good food, and by keeping in the open air a good deal, and to preventing septic infection through ulcers.

V. ZOÖNOSES.

All those infectious diseases are designated zoönoses that are usually acquired by human beings *through inoculation from animals.*

ANTHRAX.

Etiology.—Anthrax results from inoculation with **anthrax-bacilli,** which were first described by Rayer in 1851, and were subsequently carefully studied especially by Koch. The organisms are rods from 5 to 20 μ long (Fig. 83), staining readily with aniline dyes, susceptible of artificial culture and of successful inoculation upon animals. Among domestic animals anthrax occurs most frequently in cows; less commonly in horses, sheep, and swine. Other animals also, particularly herbivora, as, for instance, the stag, the deer, are, however, readily amenable to infection. In man, anthrax is most frequently acquired by *inoculation*, which occurs with especial readiness in persons that come into contact with animals suffering from anthrax, such as hostlers, veterinarians, shepherds, butchers, etc. Should blood or discharges from animals suffering from anthrax gain entrance into wounds in human beings, infection with anthrax-bacilli may readily take place. It has further been maintained that such infection may take place even through the uninjured skin. Persons also are exposed to the danger of infection who are compelled to manipulate the remains of animals dead of anthrax, as, for instance, the hair and the hide, such as tanners and furriers. There is also a possibility that anthrax is conveyed to human beings by *insects*. Such individuals are exposed to the danger of *alimentary* or *enterogenous anthrax* as use the meat, the milk, the butter, or the cheese from animals suffering from anthrax. I have observed infection also through the intestine in a physician engaged in the study of anthrax-bacilli in the laboratory, where he took breakfast, which he unconsciously infected with anthrax-bacilli. *Aërogenous* or *bronchogenous infection* with anthrax-bacilli has, among other conditions, been observed in rag-establishments, the rags or the like being contaminated with anthrax-bacilli, which are inhaled with the dust generated as a result of the manipulations to which the rags are subjected. This variety of anthrax has been designated also *rag-sorters' disease*. *Infection of one human being by another* is possible, but probably occurs only rarely. Recovery from one attack of anthrax does not confer certain protection from *subsequent attack*.

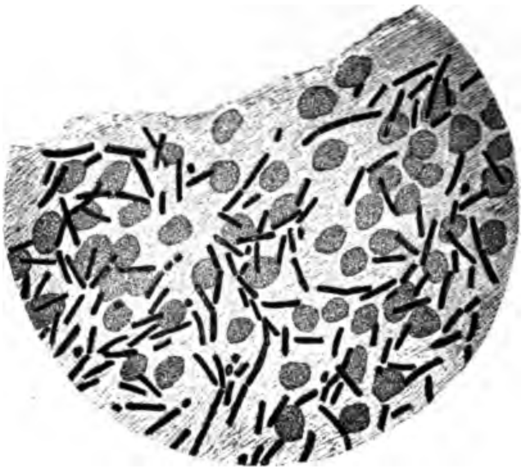


FIG. 83.—Anthrax-bacilli from the subarachnoid space of the cerebellum, from a hostler dead of anthrax-carbuncle of the left forearm; stained by Gram's method; oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

Symptoms.—The *local symptoms of anthrax* vary in accordance with the character of the infection. In cases of *inoculation-anthrax* or *cutaneous anthrax*, anthrax-carbuncle or anthrax-edema the external integument—alterations that occur at the latest a week after infection. In the presence of *anthrax-carbuncle* there develops upon the skin, with slight prickling, sticking, and burning, a hard, red nodule, over which generally the epidermis is soon raised into a flabby, wrinkled vesicle filled with serous or hemorrhagic contents. The blister ruptures within a short time, but there forms about it a circle of new vesicles of similar character, and this process may be repeated several times in the further course of the disorder. Often, **inflamed lymphatics** pass from the cutaneous alterations described to the adjacent **lymphatic glands** in the form of red cords, and the glands are generally inflamed, swollen, and painful. The diseased portions of skin frequently acquire a blackish, gangrenous appearance. *Anthrax-edema* frequently begins in the face, particularly in the eyelids. The skin is puffy, feels warm, and often exhibits a reddish erysipelatous discoloration. In places vesicular elevation of the epidermis takes place. The vesicles rupture and gangrenous alterations in the skin take place. Not rarely symptoms of anthrax-edema and anthrax-carbuncle occur in the same patient. To the local alterations of cutaneous anthrax symptoms of **general infection** become superadded in the course of about two days, particularly fever, progressive dyspnea, cyanosis, increasing coma and delirium, enlargement of the spleen, and exhaustion. Generally death occurs in the second week of the disease. *Enterogenous* or *intestinal anthrax* is attended with diarrhea, frequently with bloody stools, together with symptoms of general infection, while in cases of *aërogenous* or *pulmonary anthrax* pneumonic symptoms are most conspicuous among the local alterations. Intestinal and pulmonary anthrax may occur independently or in association with cutaneous anthrax.

Diagnosis.—The diagnosis of anthrax is generally not difficult. Frequently the history will indicate contact with animals suffering from anthrax; but, above all, anthrax-bacilli should be looked for in the inflammatory foci of the skin, or, in the event of failure to find them, inoculation-experiments on animals (guinea-pigs, rabbits, and mice) should be undertaken, and after death has occurred the viscera should be examined for anthrax-bacilli.

Prognosis.—The prognosis of anthrax is exceedingly grave. Most patients die, particularly if they come under medical treatment too late or only after the development of general infection.

Anatomic Alterations.—After death the body generally exhibits **marked rigidity** and **cyanosis**. In the internal viscera (heart, liver, kidneys, lungs, brain, etc.) **extravasations of blood** are frequently encountered. The **lymphatic glands** are increased in size and greatly distended with blood. The **spleen** and the **liver** are enlarged. Upon the intestinal mucous membrane nodules in process of breaking down are not rarely present—*intestinal anthrax-carbuncle*. In addition, the lymph-follicles of the intestinal mucous membrane and the retroperitoneal glands are enlarged. Often the **retroperitoneal connective tissue** is inflamed and edematous. On *microscopic examination,* in addition to granular turbidity and fatty degeneration of parenchymatous cells of the various viscera, collections of anthrax-bacilli are found in the blood-vessels. In the **capillaries** of some organs (lungs, kidneys, liver) these accumulations often form connected casts, which give the impression of having resulted from mechanical as well as chemic-toxic influences. Anthrax-bacilli occur also in the **lymph-spaces,** particularly those of the brain (p. 542, Fig. 83). The blood exhibits hyperleukocytosis and diminished tendency on the part of the red corpuscles to form rouleaux. In the *anthrax-carbuncle* round-cell accumulations can be recognized, with hemorrhagic infiltrations and anthrax-bacilli, the latter in part free, in part enclosed within cells. In addition, pyogenic cocci are generally present.

Treatment.—In the *prophylaxis* great care especially will be required in dealing with animals suffering from anthrax. Meat and milk and the secondary products of the latter from animals suffering from anthrax should not be used. The hide and the hair should first be sterilized before they are sold to merchants. **Excision or cauterization of an anthrax-carbuncle** would be useful only if general symptoms were not already present. Even under such circumstances many surgeons advise rather **affusions of mercuric-chlorid solution or inunctions of mercurial ointment.** Internally **stimulants** should be administered. In the treatment of intestinal anthrax **quinin, calomel,** and **intestinal infusions of saline solution** have been recommended.

GLANDERS.

Etiology.—The cause of glanders is the **bacillus of glanders** (Fig. 84) discovered by Loeffler and Schütz (1882), who succeeded in growing glanders-bacilli in pure culture and in inoculating animals successfully with them. *Glanders* occurs most frequently in horses, and all persons are exposed to the danger of infection who come in contact with animals suffering from the disease, particularly hostlers, veterinarians, and horse-slaughterers. Infection generally takes place from the entrance into open cutaneous wounds of discharges from glanders-lesions or of blood—*inoculation-glanders.* Infection is believed to be possible even through the uninjured skin. *Alimentary glanders,* induced by the use of the meat of animals suffering from glanders, probably is exceedingly rare. Whether *aërogenous infection* takes place through inhalation of the air in stables containing animals suffering from glanders, has not been demonstrated with certainty. In isolated instances *transmission from one person to another* has been observed.

Symptoms and Prognosis.—The symptoms of glanders, which develop most frequently *within from three to five days* after inoculation, resemble those of septicopyemia, and accordingly as the disease pursues a course covering from two to four or from four to twelve weeks or several months, or even years, a distinction has been made between *acute*, *subacute*, and *chronic glanders*. Chronic glanders is not rarely attended with acute exacerbations that frequently terminate fatally. Among *local alterations*, **nodules** and infiltrates occur upon the external integument, undergoing ulceration and exhibiting but slight tendency to cicatrization. Frequently, partial cicatrization takes place; but, on the other hand, the destructive process continues to extend in other situations. This fact explains the designation *worm* that is sometimes applied to glanders. The number of glanders-nodules is variable.

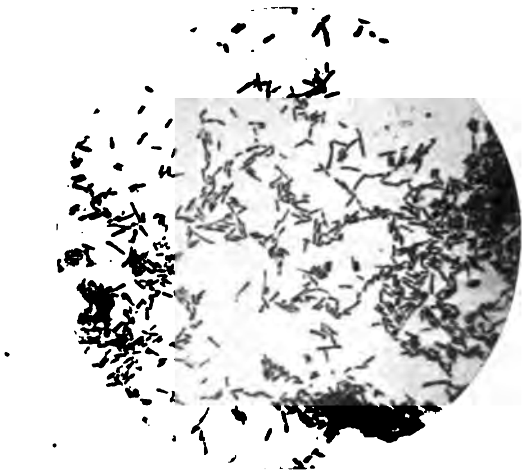


FIG. 84.—Bacillus mallei, from a culture upon glycerin agar-agar; magnified 1000 times (Fränkel and Pfeiffer).

In cases of chronic glanders new nodules continue to appear for years. In the vicinity of glanders-nodules the skin frequently exhibits erysipelatous and erythematous alterations. Inflamed lymphatics also are not rarely observed to arise from them. Adjacent **lymphatic glands** are often swollen and not rarely also undergo suppuration. Occasionally the skin is covered with large and small vesicles, suggestive of pemphigus, impetigo, and ecthyma. Frequently the **muscles** are the seat of nodules, infiltrates, and abscesses. At times **purulent arthritis** develops. Glanders-nodules and glanders-infiltrates form also upon the **mucous membranes**, subsequently undergoing purulent breaking down, and as a result giving rise to ulcers of the mucous membrane of considerable extent. The nasal mucous membrane is involved with especial frequency. Under such circumstances purulent and bloody discharges take place from the nose, and there is, in addition, pain, particularly in the frontal sinuses. The laryngeal mucous membrane likewise is frequently the seat of the lesions of glanders. Finally, abscesses form also in the **internal viscera** (lungs, myocardium, liver, kidneys, spleen, etc.) resulting from the breaking down of glanders-nodules. **General infection** is indicated by fever, which often is interrupted by chills, and by progressive loss of strength, as a result of which death generally occurs. Although recovery from glanders does take place, this is rather the exception, so that the *prognosis* is always grave.

Diagnosis.—Glanders can be recognized with certainty only by means of *bacteriologic methods*. In the first place, discharges and blood should be examined for glanders-bacilli, and in doubtful cases discharges should be introduced into the abdominal cavity of guinea-pigs, in which, in case the disease be glanders, inflammation and suppuration of the testicle soon develop. Glanders-bacilli possess readily recognizable peculiarities when cultivated upon potatoes. On the second or the third day pure cultures upon this medium form an amber-yellow coating, which in the course of eight days becomes coppery-red and surrounded by a greenish zone. The clinical picture of glanders might be confounded, in addition to that of septicopyemia, also with that of **tuberculosis** and of **syphilis**.

Anatomic Alterations.—Glanders-nodules and glanders-infiltrates consist of aggregations of round cells, which exhibit in marked degree a tendency to purulent disintegration and to caseation. They may occur in any viscus, even in cartilage and in bone.

Treatment.—In addition to **surgical treatment** of abscesses and ulcers, inunctions of **mercurial ointment** and the administration of **potassium iodid** have especially been recommended.

ACTINOMYCOSIS (RAY-FUNGUS DISEASE).

Etiology.—Actinomycosis is due to the activity of the **ray-fungus—actinomyces**—which is included among *fission-fungi*, and belongs to the variety *cladothrix*. It occurs especially upon grasses, wheat, corn, and beards. It gains entrance into the human body either from the mouth or through inhalation. Inoculation through splinters of wood upon which the ray-fungus is lodged is also known to occur. The danger of infection through carious teeth is incurred especially by such persons as indulge in the practice of introducing blades of grass between the teeth in walking in the open air.

Symptoms and Diagnosis.—The ray-fungus gives rise to purulent inflammations generally pursuing a chronic course, and exhibiting the peculiarity that **hard granules** appear in the pus secreted, and these are found on microscopic examination to contain ray-fungi. The latter exhibit in part slender filaments, in part bulbous enlargements (Fig. 85). Often chronic inflammation of the lower jaw, less commonly of the vertebral column, of other bones or of the skin, is present. Actinomycosis of the bronchi is attended with symptoms of putrid bronchitis, and similar disease of the lungs with infiltration and the formation of cavities, so that the possibility of chronic pulmonary tuberculosis is suggested. In both instances the demonstration of actinomyces-granules in the sputum would be of diagnostic significance. Often symptoms of pleuritis, peripleuritis, and mediastinitis are superadded. Intestinal actinomycosis is occasionally attended with the symptoms of perityphlitis. At times actinomyces-nodules are present in the brain, and during life they are attended with symptoms of cerebral tumor. The disease is often protracted over many years, new inflammatory and purulent foci appearing constantly, and it frequently resembles septicopyemia. A positive *diagnosis* of actinomycosis is possible only by means of bacteriologic methods.

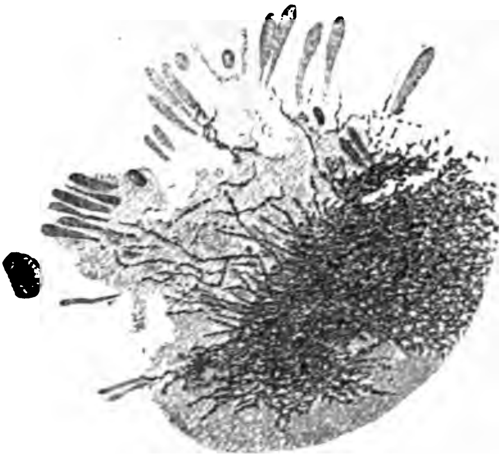


Fig. 85.—Actinomyces-granule from a disease-focus in the lung. Weigert stain; oil-immersion; magnified [illegible] times (personal observation, Zurich clinic).

The **prognosis** of actinomycosis of internal viscera is generally bad.

Treatment.—External actinomycosis has been cured in a number of instances by **operative measures**. Among internal remedies, **potassium iodid** is recommended.

FOOT-AND-MOUTH DISEASE (EPIZOÖTIC APHTHAE).

Etiology.—Foot-and-mouth disease occurs especially in *cattle*, and is acquired by human beings principally through the ingestion of unboiled or insufficiently boiled *milk* or milk-products (butter, cheese) from diseased animals. *Milkers* are occasionally infected from the entrance into wounds upon the fingers or hands, in the act of milking, of the contents of vesicles that often develop upon the udders of cows suffering from foot-and-mouth disease. The infective agent is unknown, but, in any event, it is contained, in addition to the milk, also in the blood, the saliva, the urine, and the feces.

Symptoms.—The *period of incubation* is generally three or four days. At the end of this time **fever** sets in and the temperature at times reaches the level of 40° C. (104° F.). Some two days later **vesicles** form upon the mucous membrane of the lips and tongue; less commonly upon the mucous membrane of the hard and the soft palate. In addition, vesicles appear around the nails and between the toes and the fingers, occasionally also upon the breasts. Sometimes a roseolous and erythematous exanthem appears in places. The patients complain of **burning and pain in the mouth**, while the **discharge of saliva is increased**, and occasionally they present marked gastro-enteritic symptoms, which may become dangerous especially in children, although death occurs but rarely. In the second week of the disease the cutaneous vesicles dry up into thin crusts, which soon fall off without leaving cicatrices. The disease terminates in the course of three or four weeks.

The **diagnosis** depends essentially upon a knowledge of the possibility of infection.

Treatment.—As a *prophylactic* measure the use of unboiled milk from diseased animals should be forbidden, as well as that of milk-products. Boiled milk also is rendered harmless only by *long-continued* boiling. Milkers should engage in their pursuit only if their hands and fingers are free from injury. When the disease has developed the mouth and the pharynx should be rinsed after each meal with **potassium chlorate** (5.0 : 200). Vesicles upon the skin should be covered with absorbent cotton smeared with a **borated ointment.** In the presence of gastro-enteritic symptoms **potassium chlorate** should be administered internally (5.0 : 200—75 grains : 6½ fluidounces—from 5 to 15 c.c.—from 1 to 4 fluidrams

RABIES (LYSSA; HYDROPHOBIA).

Etiology.—Human beings are generally attacked by rabies in consequence of the *bite of rabid dogs*, less commonly of other rabid animals, as, for instance, cats, horses, cows, wolves, foxes. Other modes of infection are rare, although a few years ago a pathologic anatomist at Prague became inoculated in the post-mortem examination of a dog dead from rabies, dying likewise from rabies.

The infective agent is as yet unknown, although it has been determined that it is contained in the blood and the saliva of diseased animals. Not every bite of a rabid animal is followed by rabies in man; particularly in the case of bites through the clothing the saliva of the rabid animal is often intercepted. Bites on the face, upon the hands, and upon the uncovered legs and feet, on the other hand, are especially dangerous.

Symptoms.—The *period of incubation* of rabies exhibits great variations. Generally it is from fifteen to eighty days, but occasionally it extends over six months. Reports as to periods covering several years (up to thirty) are probably beyond the limits of credulity. Pains and increased discharge from the wound are mentioned as *prodromes*, and if cicatrization has already taken place, reopening of the scar. The patient becomes pallid and restless and harassed by a fear of the terrible disease. The distinctive symptoms consist in **attacks of muscular spasm**, which at first involve the *muscles of inspiration*, and as a result cause irregular, deep, and sighing inspirations, in consequence of which respiration is greatly interfered with. The patient at the same time becomes cyanotic. Soon *spasm in deglutition* appears, which at first is manifested only on attempts at swallowing, but subsequently sets in as soon as food and drink are brought into view, and even upon the thought of them, thus rendering the ingestion of food impossible. Such spasms occur also at the sight of water, and the disease has, therefore, been designated *hydrophobia*. From time to time, subsequently, attacks occur, attended with delirium, snapping movements of the jaws, and also **general convulsions**. These attacks follow one another with gradually increasing frequency, lasting longer and longer, and frequently causing death amid progressive exhaustion, at the latest at the end of the second week of the disease.

Diagnosis.—The diagnosis of rabies is generally easy, particularly if the history renders certain antecedent infection. In doubtful cases portions of the medulla oblongata of those dead of the disease may be injected beneath the dura in rabbits, and the development of morbid symptoms in the animals awaited.

Prognosis.—The prognosis was formerly serious, but since the discovery of curative inoculation by Pasteur most patients are saved.

Anatomic Alterations.—Anatomic alterations distinctive of rabies are not known. Hemorrhages into the meninges and into the substance of the brain and the spinal cord have been described as secondary conditions. On microscopic examination round-cell accumulation, swelling and vacuolation in the ganglion-cells, and swelling of nerve-fibers have been observed in the central nervous system.

Treatment.—A high license for dogs, the constant wearing of muzzles, and the leading of dogs with a chain may be recommended as *prophylactic* measures for the prevention of rabies in dogs. Bite-wounds should at once be sucked and be cauterized with the **hot iron** or with **potassium hydroxid.** The patient should besides be sent as speedily as possible to an institution for the practice of *inoculation against rabies*, such as has been erected in Paris, St. Petersburg, Vienna, and Berlin, in order to be subjected to **curative inoculations** according to the method of Pasteur. If rabies has developed in an individual, **chloral hydrate** (5.0—75 grains) should be administered and **inhalations of chloroform** practised if muscular spasm appears.

INDEX TO VOLUME II.

ABASIA, hysterical, 143
Aboulia in hysteria, 143
Acanthosis nigricans, 202
Acarus folliculorum, 216
  scabiei, 211
Achorion Schönleinii, 222
Acidophile cells, 239
Acne, cachectic, 181
  disseminated, 180
  frontal, 181
  hordeolar, 181
  indurated, 181
  mentagra, 182
  necrotic, 181
  punctate, 181
  pustular, 181
  rosacea, 184
  syphilitic, 181, 515
  toxic, 181
  urticate, 181
  varioliform, 181
  vulgaris, 180
Acrodynia, 169
Acroparesthesia, 120
Actinomycosis, 546
Agraphia, 41
  amnesic, 42
  motor, 42
  sensory, 42
Akinesis algera, 143, 147
Akromegaly, 136
  partial, 138
Albinism, 204
Alexia, 41
Alkaptonuria, 299
Alopecia, 205
  celsi, 206
  circumscribed, 206
  syphilitic, 515
American disease, 145
Amimia, 41
  amnesic, 42
  motor, 42
  sensory, 42
Amœba coli, 403
Anal tenesmus, 405
Anarthria, 19
Anemia, progressive pernicious, 250
Anemia, progressive pernicious, blood in, 251
    circulatory symptoms in, 253
    diagnosis, 257
    hemorrhages in, 254
    nature of, 257
    nervous system in, 256
    treatment, 257
Anemic fever, 253
Angina, herpetic, 337
  Ludwig's, 460
Angioneurotic edema, intermittent, 121
Anidrosis, 194
Anthrax, 541
  bacillus of, 541
  carbuncle of, 543
  edema of, 543
  treatment, 544
Antidiphtheric serum, 462, 469
Antitoxin, tetanus, 425
Anus, eczema of, 172
Aphasia, 39
  amnesic, 39, 40
  ataxic, 39, 44
  cerebral hemiplegia and, 46
  conduction-, 44, 45
  congenital, 47
  cortical, 43
  diagram, 44
  doctrine of, 43
  mixed, 40
  motor, 39
    cortical, 44
  optic, 40
  sensory, 33, 39, 40
  simple, 40
  spastic, 94
  subcortical, 44
    motor, 45
    sensory, 44
  transcortical, 44
    motor, 44
    sensory, 44
  treatment, 47
Apoplectic attack, 55
    restoration of consciousness after, 56

Apraxia, 43
Arachnoid hemorrhage, 84
Arteries, gummata of, 529
Arthritic tophi, 281
Arthritis deformans, 308
  gonorrheal, 426
  of poor, 308
Asiatic cholera, 409
    abdomen in, 415, 417
    anomalies, 415
    bacillus of, 409
    diagnosis, 416
    epidemics of, 412
    fencing attitudes in, 417
    intestines in, 417
    kidneys in, 418
    media of infection, 409–412
    spleen in, 417
    stool of, 413
    symptoms, 413
    treatment, 418
    vomitus of, 414
Asses' cough, 375
Association-fibers, 34
Astasia, hysterical, 143
Athetosis, 106
Atrichia, 205
Atrophy, muscular infantile, 157
    juvenile, 155
    neural progressive. 158
    progressive myopathic, 153
Aura, epileptic, 88

BACILLUS, comma, 409
  Löffler, 456, 470
  mallei, 545
  of anthrax, 541
  of diphtheria, 456, 470
  of influenza, 380
  of leprosy, 537, 538
  of tetanus, 449
  plague, 372
  tubercle. See *Tubercle bacillus.*
  typhosus, 388
Back, atrophy and weakness of, 155
Banting's treatment of obesity, 278
Barlow's disease, 269
Bartholinitis, gonorrheal, 430
Basophile cells, 239
Beard, eczema of, 172
  herpes tonsurans of, 227
Bedbug, 220
Bergeron's electric chorea, 107
Black death, 371
Bladder, tuberculosis of, 491
    treatment, 495
Bleeders' disease, 270
Blindness, mind-, 34
  word-, 34
Blood, diseases of, 238
  dry preparations of, 239
Blood, number of colorless corpuscles in, determination of, 240
Blood-sedimenting method, Walker's, 240
Blood-serum reaction of Widal, 388
Blood-sweating, 142, 195
Blue cough, 376
Bone-marrow, involvement of, in leukemia, 243
  lymphoid, 245
  pyoid, 245
  red, 245
Bones, rachitis of, 300
  tertiary syphilis of, 523
Brain, abscess of, 63
    encapsulated, 64
    free, 64
    from otitis interna, 64
    latent, 65
    treatment, 66
  anemia of, 47
  arteries at base of, distribution, 27
  atrophy of, 78
  base of, disease of, focal symptoms in, 39
  convolutions of convexity of, 29
  diffuse sclerosis of, 78
  diseases of, 29
    general symptoms in, 30
    local symptoms in, 30
  edema of, 52
  hemiatrophy of, infantile, 78
  hemorrhage of, 52. See also *Encephalorrhagia.*
  hyperemia of, 50
  hypertrophy of, 77
  inflammation of, 63. See also *Encephalitis.*
  necrotic softening of, 60
  parasites of, 72
  sinuses of, inflammation of, 79
      treatment, 82
    thrombosis of, 79
      treatment, 82
  syphilis of, 530
  tumors of, 68
    latent, 69
    papillitis and, 70
Breast, eczema of, 172
Bromidrosis, 195
Bronchi, tertiary syphilis of, 525
Bubo, 439
  in syphilis, 513
Bubonic plague, 371
    bacillus of, 372
    treatment, 374
Bulbar monoplegia, 17
  myelitis, acute, 28
  paralysis, acute, 25
    apoplectic, 25
    chronic progressive, 20

Bulbar [illegible]
Bur[illegible]

[illegible]

Chorea, reflex, 101
  senile, 100
  symptomatic, 104
  toxic, 101
  treatment, 104
Chromidrosis, 195
Chvostek's facial phenomenon, 97
Cimex lectularius, 220
Circulatory organs, tertiary syphilis of, 528
Clothing-louse, 219
Colles' law, 532
Color-analytical blood-examination, 239
Colotyphoid, 399
Coma, diabetic, 292
Comedo, 198
Comma bacillus, 409
Commissural fibers, 34
Conjunctiva in small-pox, 345
Connective tissue, induration of, in newborn, 203
    subcutaneous, cysticercus of, 220
Consumption, galloping, 480
  pulmonary, 470. See also *Tuberculosis*.
Continued fever, 368
Corona radiata, 34
Corpus callosum, disease of, 39
Corpuscles, colorless, in blood, determination of number of, 240
  red, in leukemia, 241
Cough, croupy, 465
Cowper's gland, gonorrheal inflammation of, 426
Crab-louse, 218
Cramp, choleraic, 415
  writers', 98
Cranium, lesions of, 39
Cretinism, sporadic, 128
Croup, laryngeal, 464
Cutaneous neuroses, 209
  sand, 198
Cutis, atrophy of, 208
  testea, 197
Cysticercus cellulosæ in brain, 72
  of subcutaneous connective tissue, 220
Cystinuria, 299

DEAFNESS, word-, 39, 49
Deferentitis, gonorrheal, 425
Defluvium capillorum, 206
Dermatitides, 164
  bullous, 176
  erythematous, 164
  papular, 190
  pustular, 179
  squamous, 186
  vesicular, 170
Dermatitis, contusiform, 167
  exfoliative, 178
Dermatomycoses, 221
Dermatoses, parasitic, 211
Dermatozoönoses, 211
Diabetes insipidus, 296
    and mellitus, 297
    urine in, 297
  mellitus, 285
    and insipidus, 297
    coma in, 292
    complications, 291
    deceptive, 287
    diagnosis, 293
    diet in, 295
    idiopathic, 286
    intermittent, 293
    lungs in, 291
    nervous disorders in, 292
    pathogenesis, 294
    quantitative determination of sugar in urine in, 289
    stomach-dilatation in, 291
    symptomatic, 286
    tests for sugar in urine in, 287
    treatment, 294
    urine in, 287–290
Diabetic coma, 292
Diarrhea, cholera-, 413
Diathesis, uric acid, 279. See also *Gout*.
Digestive organs, tertiary syphilis of, 526
Diphtheria, 456
  antitoxin for, 462
  bacilli of, 456, 470
  laryngeal, 464
    diagnosis, 467
    serum for, 469
    suffocation in, 466
    treatment, 469
  nasal, 470
  of nasal mucous membranes, 459
  pharyngeal, 456
    diagnosis, 461
    heart in, 462
    nervous affections and, 460
    paralysis with, 460
    serum for, 462
    treatment, 462
    varieties, 458, 459
  scarlatinal, 328
Dubini's electric chorea, 107
Dura mater, cerebral, hematomata of, 83
Dysentery, 403
  adynamic, 406
  amebic, 403
  catarrhal, 404
  diagnosis, 408
  follicular, 404
  liver-abscess in, 407
  metastases of, 407
  necrotic, 404
  peritonitis with, 407

Dysentery, polyarthritis in, 407
  purulent, 404
  putrid, 406
  septic, 406
  stool of, 406
  treatment, 408

EAR, eczema of, 172
Ebstein's treatment of obesity, 278
Echinococci in brain, 73
Echokinesis, 108
Eclampsia, 93
Ecthyma, 179
  syphilitic, 515
Eczema, 170
  marginate, 227
  papular, 170
  treatment, 174
Edema, angioneurotic, intermittent, 121
Ehrlich's color-analytical blood-examination, 239
Elastic fibers in sputum in tuberculosis, 476
Encephalitis, 63
  congenital, 68
  hemorrhagic, 66
Encephalomalacia, inflammatory, 66
Encephalorrhagia, 52
  amount of blood extravasated in, 54
  dementia in, 58
  diagnosis, 58
  stages, 55
  symptoms, 55–58
  treatment, 59
English sweating-sickness, 338
Enteric fever, 387. See also *Typhoid fever.*
Eosinophile cells, 239
Ephelides, 199
Epidemic cerebrospinal meningitis, 442. See also *Meningitis*
Epilepsy, toxic, 87
  treatment, 93
  true, 87
Epileptic attack, 89
  aura, 88
  equivalents, 91
Epileptoid states, 91
Epizoötic aphthæ, 548
Erysipelas, 329
  afebrile, 332
  bullous, 332
  cutaneous, 332
  gangrenous, 333
  of mucous membranes, 333
  pustular, 332
  treatment, 333
  vesicular, 332
  wandering, 333
Erythema, annular, 168
  gyrate, 168
  iris, 168
  mammellatum, 168
  multiform exudative, 168
  nodose, 166
  recurring scarlatiniform, 169
  syphilitic, 514
Erythrasmia, 229
Erythromelalgia, 121
Esophagus, mucous membrane of, in small-pox, 345
  muscles of, paralysis of, 23
  tertiary syphilis of, 527
European cholera, 420
Exanthemata, acute infectious, 311
  syphilitic, 514
Exophthalmic goiter, 131
    treatment, 134
Exophthalmos, 132
Extremities, eczema of, 173

FACE, eczema of, 171

Flexibility of muscles, waxy, 114
Foot-and-mouth disease, 548
Fortification-scotomata, 119
Freckles, 99

GANGRENE, symmetrical, 122
Garrod's thread-test, 282
Genitalia, eczema of, 172
  seborrhea of, 196
Genito-urinary organs, tertiary syphilis of, 528
Gerlier's disease, 113
Giant-growth, morbid, 136
  partial, 138
Gingivitis, scorbutic, 266, 267
Glanders, 544
Globus, hysterical, 140
Glossitis, variolous, 344
Glossopharyngeal nucleus, situation, 20
Glottis, spasm of, tetany and, 97
Gluteal muscles, pseudohypertrophy of, 155
Glycosuria, alimentary, 294
  hematogenous, 294
  myogenous, 294
  nephrogenous, 294
  neurogenous, 294
Goiter, exophthalmic, 131
    treatment, 134
Gonococci, 421
Gonorrhea, 421
  acute, in female, 430
      treatment, 436
    in male, 423
      treatment, 433
  chronic, in female, 431
      treatment, 436
    in male, 428
      treatment, 435
  diagnosis, 423
  gonococci of, 421
  gouty, 427
  in children, 422
  morning-drop in, 428
  treatment, 433
  two-glass test in, 429
  urethral injections in, 433
Gonorrheal Bartholinitis, 430
  metastases, 424, 426
  threads, 428
Gout, 279
  heart in, 283
  kidneys in, 283
  nature of, 284
  treatment, 284
Gouty deposits, explanation, 284
  nodules, 281
  ulcers, 282
Graefe symptom, 133
Graves' disease, 131
    treatment, 134
Grip, 380. See also *Influenza.*
Gummata, 522–532

HAFFKINE'S prophylaxis, 374
Hairs, atrophic changes in, 205
  brittleness of, 207
  deficiency of, 205
  graying of, 205
  hypertrophy of, 202
  loss of, 205
Hair-spleen, 234
Hand, palm of, eczema of, 173
  sweating of, 194
Headache, habitual, 115
  nervous, 115
Head-louse, 216
Heart, fat, in obesity, 277
Heberden's nodes, 308
Hectic fever, 480
  flush, 480
Hematuria, winter, 272
Hemianesthesia, hysteric, 141
Hemianopsia, 33
Hemiatrophy, progressive facial, 123
Hemichorea, 103
Hemicrania, 117
  alternating, 118
  ophthalmic, 119
  sympathetic-paralytic, 119
  sympathetic spastic, 119
  treatment, 120
  vasomotor, 119
Hemicranic equivalent, 120
Hemidrosis, 142, 195
Hemiplegia, crossed, 17
Hemihypertrophy, facial, 125
Hemiplegia, alternating, 25
  cerebral, 36
  cortical, development, 32
Hemoglobinemia, 272
Hemoglobinuria, paroxysmal, 272
Hemophilia, 270
Herpes, progenital, 337
  tonsurans, 226
  zoster, 334
    neuralgia with, 335
Hirsuties, 202
Hives, 164
Huntington's chorea, 105
Hutchinson's teeth, 534
Hydrocephalus, 74
  acquired, 75
  acute, 76
  cachectic, 75
  chronic, 76
  circumscribed, 76
  congenital, 74
  external, 76
  intermeningeal, 76
  internal, 76
  ventricular, 76

Hydrophobia, 549
Hyperidrosis, 193
Hyperthyroidism, 134
Hypertrophy, muscular, true, 159
    with juvenile atrophy, 156
  pseudomuscular, 153, 158
Hypoglossal nucleus, situation, 20
Hysteria, 139
  diagnosis, 144
  eye in, 142
  hearing in, 142
  paralysis with, 140
  treatment, 144
Hysterical globus, 140
Hystero-epilepsy, 143

ICHTHYOSIS, 200
  sebaceous, 197
Impetigo, 179
  syphilitic, 515
Infectious diseases, 311
    of typical localization, 311
    of varying localization, 456
    with changes in blood, 357
      in blood-generating organs, 357
      in digestive organs, 384
      in joints and muscles, 349
      in nervous system, 442
      in respiratory organs, 374
    with lesions of sexual organs, 421
Influenza, 380
  abdominal, 383
  bacillus of, 380
  pneumonia with, 382
  treatment, 383
Insanity, pre-epileptic, 89
Intermittent fever, 364
    comatose, 367
    duplicated, 367
    larval, 367
    pernicious, 367
    quotidian, 364-366
    tertian, 366
    treatment, 371
Internal capsule, disease of, focal symptoms in, 36
Intestine, tertiary syphilis of, 527
  tuberculosis of, 488
  tuberculous ulcers of, 190
Itch, 211
  treatment, 215
Itch-burrow, 214
Itch-parasite, 212
Itching weeping tetter, 170

JACKSONIAN epilepsy, 33, 87
Joints, dropsy of, intermittent, 121
  infectious diseases with changes in, 349
  tertiary syphilis of, 524

KEEL-BREAST, 302
Keratosis, 200
Kidneys, pelvis of, tuberculosis of, 492
  tuberculosis of, 491
    treatment, 495

LAGOPHTHALMOS, choleraic, 414
Laryngeal mucous membrane in small-pox, 345
Laryngitis, herpetic, 337
Larynx, consumption of, 486
  phthisis of, 486
  tertiary syphilis of, 525
Legs, eczema of, 173
Lentigenes, 199
Leontiasis ossea, 138
Leprosy, 537
  bacillus of, 537, 538
  cutaneous, 539
  diagnosis, 540
  facies in, 539
  nervous, 540
  treatment, 541
  ulcers of, 539
Leprous leontiasis, 539
  satyriasis, 539
Leukemia, 238
  blood in, 239
  colorless corpuscles in, 239
  complications, 244
  diagnosis, 245
  involvement of bone-marrow in, 243
  lymphatic, 243
  nature of, 246
  onset, 243
  red corpuscles in, 241
  spleen in, 242
  splenic enlargement in, 242
  treatment, 246
Leukoderma, 204
  syphilitic, 515
Lice, 216
  clothing, 219
  crab, 218
  head, 216
Lichen of scrofulous, 191
  ruber, 191
    planus, 192
  syphilitic, 515
Lips, eczema of, 172
Liver, fatty, in obesity, 277
  syphilitic lobulated, 528
  tertiary syphilis of, 527
Lock-jaw, 452
Löffler bacillus, 456, 470
Ludwig's angina, 460
Lues, 510. See also *Syphilis.*
Lumbago, 356
Lungs, tertiary syphilis of, 526
  tuberculosis of, 470. See also *Tuberculosis, chronic pulmonary.*

Lymphatic glands, scrofulous enlargement of, 506
Lyssa, 549

MALARIAL cachexia, 368
  fever, 361
    diagnosis, 364
    melanemia and, 368
    mosquitoes and, 364
    plasmodia of, 362
    symptoms, 364
    treatment, 369
Mammary gland, gummata of, 526
Mange, 212
Mast-cells, 240
Measles, 316
  anomalies of, 319
  diagnosis, 320
  eruption of, 318
  laryngeal complications, 319
  otitis media in, 320
  sequelæ, 320
  temperature-curve, 317
  treatment, 321
Measles-pneumonia, 320
Medulla oblongata, acute inflammation of, 28
    arteries of, embolism of, 26
      thrombosis of, 26
    diseases of, 17
    hemorrhage into, 25
    neoplasms of, 28
    transverse section through, 21
Megaloblasts, 252
Melanemia, 368
Melanoderma, syphilitic, 515
Ménière's disease, 113
Meninges, cerebral, diseases of, 79
    gummata of, 530
    hemorrhage into, 84
Meningitic spots, 444
Meningitis, abortive, 445
  apoplectiform, 445
  cerebrospinal, epidemic, 442
      anatomic changes, 446
      diagnosis, 446
      treatment, 447
    secondary purulent, 448
    serous, 449
    tuberculous, 498
      abdomen in, 501
      diagnosis, 502
      respiration in, 501
      spinal cord in, 499
      symptoms, 500
      treatment, 502
  fulminating, 445
  intermittent, 445
Meningococcus intracellularis, 442
Meningotyphoid, 394
Mercurialism, 520

Metabolism, disorders of, 275
Metastases, gonorrheal, 424, 426
Methylene-blue for malaria, 370
Miasm, 362
Microsporon furfur, 221
Migrain, 117. See also *Hemicrania*
Migrainous status, 118
Miliaria, 175
Miliary fever, 338
  tuberculosis, 495
Milium, 198
Mind-blindness, 34
Mogigraphia, 98
Moist tetter, 170
Monophasia, 40
Monoplegia, 32
  bulbar, 17
Moore's test for sugar in urine, 287
Morbilli, 316. See also *Measles.*
Morbus maculosus Werlhofii, 264
Morning-drop, 428
Mosquitoes and malaria, 364
Mother-mark, 199
Motor cortical centers, 30, 31
Movable spleen, 236
Mumps, 384
  orchitis in, 386
Muscles, diseases of, 153
  infectious diseases with changes in, 349
  tertiary syphilis of, 523
Myasthenia, 163
Myelitis, bulbar, acute, 28
Myelocytes, 240
Myocardium, tertiary syphilis of, 528
Myoclonus, 107
Myositis, progressive ossifying, 160
Myotonia, 162
Myxedema, 128, 138
Myxœdema congenitum, 130
  strumiprivum, 129

NAILS, atrophic disorders of, 208
  favus of, 225
  hypertrophy of, 203
Narco-epilepsy, 92
Nephritis, scarlatinal, 328
Nervous disorders, functional, 86
  fever, 394
  system, diseases of, 17
    tertiary syphilis of, 529
Nettle-fever, 164
Nettle-rash, 164
Neurasthenia, 144
  diagnosis, 148
  treatment, 148
Neuron, cerebrospinal, 82
Neuroses, central, 86
    with motor disturbances predominant, 87
    with psychic alterations predominant, 139

Neuroses, central, with sensory disturbances predominant, 115
  with trophic disturbances predominant, 120
  with vasomotor disturbances predominant, 120
 cutaneous, 209
 occupation-, 98
 traumatic, 149
  spinal symptoms, 150
  treatment, 152
 vasomotor articular, intermittent, 121
  of extremities, 120
Neutrophile cells, 240
Nevus, 199
Nigrities, 219
 syphilitic, 515
Nodes, Heberden's, 308
Nodose erythema, 166
Nodules, gouty, 281
Normoblasts, 252
Nose, eczema of, 172
 tertiary syphilis of, 524
Nucha, eczema of, 172
Nylander's test for sugar in urine, 288

OBERMEIER's spirilla, 358
Obesity, 275
 drinking-cures for, 279
 treatment, 278
Occupation-neuroses, 98
Oertel's treatment of obesity, 278
Oïdium Schönleinii, 222
Ointment, variolous, 347
Oligosteatosis, 197
Onychauxis, 203
Onychomycosis favosa, 225
Ophthalmoplegia, chronic progressive, 25
 external nuclear, 25
Optic thalamus, disease of, 38
Orchitis in mumps, 386
Osmidrosis, 193, 195
Osteitis, deforming, 138
Osteo-arthropathy, hypertrophic, 138
Osteomalacia, 305
Osteopsathyrosis, syphilitic, 524
Oxaluria, 299

PACHYMENINGITIS, cerebral, internal hemorrhagic, 82
Palate, soft, paralysis of muscles of, 22
 tertiary syphilis of, 526
Pancreas, tertiary syphilis of, 528
Papillitis and brain tumors, 70
Paralysis, acute cerebral, of childhood, 67
 agitans, 108
  pen-holder position in, 111
  treatment, 111
 bulbar. See *Bulbar paralysis.*
Paralysis of cerebral nerves, syphilis and, 531
 of cervical sympathetic, 87
 of depressor of epiglottis, 22
 of muscles of esophagus, 23
  of soft palate, 22
  of vocal band, 23
 pseudobulbar, 24
Paramyoclonus multiplex, 107
Paraphasia, 40, 44, 45
Parasteatosis, 198
Paridrosis, 195
Parotitis, epidemic, 384
  orchitis in, 386
Pectus carinatum, 302
 gallinaceum, 302
Pediatrophy, syphilitic, 533
Pediculi, 216
Pediculi-salve, 218
Pediculus capitis, 216
 pubis, 218
 vestimentorum, 219
Pellagra, 169
Pemphigus, 176
 acute, of newborn, 176
 syphilitic, 515
Pen-holder position, 111
Penis, chorda, 424
Pericarditis, tuberculous, 505
Peripheral nerves, tertiary syphilis of, 529
Peri-splenitis, 233
Peritoneum, tertiary syphilis of, 523
Peritonitis, tuberculous, 502
  abdomen in, 503, 504
Pernicious anemia, progressive, 250. See also *Anemia.*
Pertussis, 374
 diagnosis, 378
 in pregnancy, 378
 larynx in, 376
 stomach in, 377
 trachea in, 376
 treatment, 379
Pertussis-pneumonia, 378
Pharyngitis, acute phlegmonous, 387
Pharynx, chronic tuberculosis of, 488
 tertiary syphilis of, 526
[illegible], treatment, 197
Phosphaturia, 299
Phthisical habitus, 471, 478
  treatment, 485
Phthisis, florid pulmonary, 480
 pulmonary, 470. See also *Tuberculosis.*
Piedra, 208
Pituitary body, hyperplasia of, 69
 struma, 69
Pityriasis, rubra, 189
 simplex, 197
 versicolor, 221
Plague, 371

Plague, bacillus of, 372
  treatment, 374
Plague-pneumonia, 373
Plasmodia of malaria, 362
Pleura, tertiary syphilis of, 526
Pleuritis, tuberculous, 505
Plica polonica, 171, 217
Pneumonia, gelatinous, 474
  measles-, 320
  pertussis-, 378
  plague-, 373
  white, 535
  with influenza, 382
Pneumotyphoid, 394
Polio-encephalitis, acute infantile, 67
  progressive inferior, 20
    facial expression in, 22
    treatment, 24
  superior, 24
Polyarthritis, acute primary, 349
    diagnosis, 352
    treatment, 352
  deforming, 138
  scarlatinal, 328
Polychromophilia, 252
Polymyositis, 161
Polysarcia, 275
  treatment, 278
Polyuria, 296
Pons, disease of, focal symptoms in, 37
  Varolii, disease of, 37
Pontine symptoms, 38
Porcupine-people, 200
Porencephaly, 68
Post-epileptic state, 90
Pre-epileptic insanity, 89
Pregnancy, pertussis in, 378
  typhoid fever in, 390
Preparoxysmal insanity, 89
Profeta's law, 533
Prostatitis, gonorrheal, 426
Prurigo, 190
  buboes, 190
  urticaria with, 190
Pruritus, cutaneous, 209
Pseudobulbar paralysis, 24
Pseudoleukemia, 247
  treatment, 250
Pseudotabes, post-diphtheric, 461
Psoriasis, 186
  syphilitic, 515
Pulex irritans, 220
  penetrans, 220
Pulices, 220
Pulmonary tuberculosis, chronic, 470.
Purpura, 262
  fulminant, 264
  hemorrhagic, 264
  rheumatic, 264
  simple, 263
Pryamidal cells of cerebral cortex, 32

Quadrigeminate bodies, lesions in, 38

Rabies, 549
Rachitic rosary, 302
Rachitis, 299
  chest in, 301
  clavicles in, 302
  diagnosis, 303
  epiphyses in, 302, 303
  of bones, 300, 304
  of pelvic bones, 302
  pathogenesis, 304
  scapulæ in, 302
  sternum in, 302
  teeth in, 301
  treatment, 305
Rag-sorters' disease, 542
Ray-fungus, 546
Raynaud's disease, 122
Relapsing fever, 357
  abscess of spleen in, 360
  bilious, 360
  perisplenitis in, 360
  spirilla of, 358
  temperature-curve of, 359
  treatment, 361
Remittent fever, 368
Renotyphoid, 394
Respiratory organs, tertiary syphilis of, 524
Rheumatism, acute articular, 349
    diagnosis, 352
    treatment, 352
  chronic articular, 354
  muscular, 356
Rhinophyma, 184
Roseola, syphilitic, 514
Rötheln, 322
Rubeola, 322
Rupia, syphilitic, 523

Sago-spleen, 234
Saline discharge, 173
Saltatory spasm, 107
Sand, cutaneous, 198
Sarcocele, syphilitic, 528
Sarcoptes hominis, 211
Scabies, 211
  Norwegian, 215
  treatment, 215
Scalp, eczema of, 171
  herpes tonsurans of, 227
  seborrhea of, 195
Scarlet fever, 323
    anomalies, 326
    complications, 327
    diagnosis, 329
    eruption of, 325
    febrile albuminuria in, 328
    temperature-curve, 325

Scarlet fever, tongue in, 326
  treatment, 329
Schistocytes, 252
Sclerema of newborn, 203
Sclerodactyly, 126
Scleroderma, 126
Scorbutic gingivitis, 266, 267
Scorbutus, 266
  hemorrhages in, 267
Scotomata, fortification-, 119
Scriveners' palsy, 98
Scrofulosis, 506
  erethetic, 507
  facies in, 507
  torpid, 507
  treatment, 509
Scrofulous habitus, 507
  lichen of, 191
Scurvy, 266
  hemorrhages in, 267
Sea-scurvy, 266
Sebaceous glands, secretory disorders of, 195
  secretion, diminished, 197
    disorders of, 198
    increased, 195
Seborrhea, 195
  universal, 197
Semi-ovale, centrum, disease of, focal symptoms in, 34
Serum, antidiphtheric, 462, 469
  tetanus, 455
Shaking palsy, 108
Shock and traumatic neuroses, 149
Skin, angioneurosis of, 166
  atrophy of, 204
  discolorations of, 200
  diseases of, 164
  erysipelas of, 332
  hemorrhage into, 262
  herpes tonsurans of, 227
    zoster of, 335
  hypertrophies of, 199
  inflammations of, 164
    bullous, 176
    erythematous, 164
    papular, 190
    pustular, 179
    scaly, 186
    vesicular, 170
  itching of, 209
  parasites of, 211
    animal, 211
    vegetable, 221
  pigment of, atrophy of, 204
    hypertrophies of, 199
  secretory disorders of, 193
  tertiary syphilis of, 522
Skull, floor of, 41
Small-pox, 341
  abortive, 346
Small-pox, confluent, 346
  conjunctiva in, 345
  diagnosis, 346
  eruption of, 343
  esophagus in, 345
  hemorrhagic, 346
  immunity from, 342, 348
  pustule of, 347
  stomach in, 345
  temperature in, 345
  temperature-curve, 343
  treatment, 348
Spanish collar, 424
Spasmodic tic, 108
Speech, acquisition of, diagrams, 43
  disorders of, 43
Spermatocystitis, gonorrheal, 426
Spinal bulb, 17
  cord, syphilis of, 529
  irritation, 146
Spirilla of relapsing fever, 358
Spleen, abscess of, 231
  amyloid, 234
  capsule of, inflammation of, 233
  carcinoma of, 235
  diseases of, 230
  displacement of, 237
  echinococcus of, 235
  embolic infarction of, 230
  ham-, 234
  in leukemia, 242
  movable, 236
  rupture of, 236
  sago-, 234
  sarcoma of, 235
  tertiary syphilis of, 528
  wandering, 236
Splenic artery, aneurysm of, 238
Splenitis, 233
Sporadic cretinism, 128
Sputa globosa fundum appetentia, 478
Sputum, elastic fibers in, in tuberculosis, 476
  globose, 478
  in tuberculosis, 477
  nummular, 478
  tubercle-bacilli in, 475
Status epilepticus, 90
Stellwag's symptom, 133
Sterility after gonorrheal urethritis in male, 425
Stomach in small-pox, 345
  tertiary syphilis of, 527
Strawberry tongue, 326
Streptococcus erysipelatosus in skin, 331
Striate body, 36
Stroke, 55
St. Vitus' dance, 100
    diagnosis, 104
    treatment, 104
Subarachnoid hemorrhage, 84

Subdural hemorrhage, 84
Substantia nigra, 36
Subsultus tendinum in typhoid, 394
Sugar in urine, quantitative determination, 289
    tests for, 287
Superior longitudinal sinus, thrombosis of, 81
Swallowing, disorders of, 19
Swamp-fever, 361. See also *Malarial fever*.
Sweat, alterations in, 195
  diminished secretion of, 194
  increased secretion of, 193
Sweat-glands, secretory disorders of, 193
Sweating, axillary, 193
  blood-, 142, 195
  of feet, 194
  of hands, 194
  sickness, English, 338
  unilateral, 193
Sycosis, 182
Sydenham's chorea, 100
    diagnosis, 104
    treatment, 104
Sylvian fossa, artery of, embolism of, 62
Sympathetic, cervical, irritation of, 86
    paralysis of, 85
  nerve, diseases of, 85
Syphilids, 511, 514
  diagnosis, 517
  in hereditary syphilis, 534
Syphilis, 510
  acquired, 510
    diagnosis, 517
    treatment, 518
  alopecia in, 515
  broad condylomata in, 514, 516
  buboes in, 513
  diagnosis, 517
  hemorrhagic, 518
  hereditary, 532
    anatomic changes, 535
    Hutchinson's teeth in, 534
    late, 533
    syphilids in, 534
    tertiary symptoms in, 534
    treatment, 536
  infection with, 510, 511
  lichen of, 515
  malignant, 518
  mercurialism and, 520
  mucous membranes in, 516
  primary, 510
    treatment, 518
Syphilis, tertiary, treatment, 521, 522
  treatment, 518
    inunction, 519
  vaccination and, 511
  visceral stage, 516

TABES dorsalis, syphilis and, 529
  mesenteric, 508
Tegmentum, 36
  disease of, 37
Tendon-sheaths, gout of, 281
Tenesmus, anal, 405
Test, fermentation-, for sugar, 288
  for sugar in urine, 287
  Moore's, for sugar, 287
  Nylander's, for sugar, 288
  Trommer's, for sugar, 288
  two-glass, 429
Testicle, inflammation of, in mumps, 386
  tertiary syphilis of, 528
Tetanus, 449
  antitoxin for, 455
  bacillus of, 449
  diagnosis, 454
  facies in, 453
  localized, 454
  serum for, 455
  treatment, 454
Tetany, 94
  and laryngismus stridulus, 97
  Chvostek's facial phenomenon in, 97
  thyroid gland and, 95
  Trousseau phenomenon in, 97
Tetter, moist, 170
Thalamus, optic, disease of, 38
Thigh, pseudohypertrophy of extensors of, 155
Thomsen's disease, 162
Thorax, paralytic, 479
  permanent expiratory, 479
Thread-test, Garrod's, 282
Thrombophlebitis, treatment, 82
Tongue, strawberry-, 326
  tertiary syphilis of, 526
Tophi, arthritic, 281
Trachea, tertiary syphilis of, 525
Transverse sinus, thrombosis of, 81
Tremor, 112
Trichophyton onychomycosis, 228
Trichophyton-fungi, 227
Trichorrhexis, 207
Trismus, 452
Trommer's test for sugar in urine, 288
Trousseau phenomenon in tetany, 97
Tubercle bacillus in sputum, 475
    in urine, 493

Tuberculosis, chronic, of bladder, 491
    treatment, 495
  of kidneys, 491
    treatment, 495
  pharyngeal, 488
  pulmonary, 470
    auscultatory phenomena in, 480
    complications, 481
    diagnosis, 475
    elastic fibers in, 476
    examination of lungs in, 479
    night-sweats in, 480
    pneumothorax in, 481
    sputum in, 477
    stomach in, 480
    symptoms, 475
    treatment, 483
  general miliary, 495
    treatment, 498
  of pelvis of kidney, 492
  solitary, 510
  urogenital, 491
Tuberculous caverns, 474
  ulcers of intestine, 490
Two-glass test, 429
Typhoid, bilious, 360
  cholera-, 416
  fever, 387
    abdomen in, 392
    abortive, 393
    afebrile, 394
    ambulatory, 394
    bacilli of, 388
    bowels in, 393
    constipation in, 396
    course, 391
    diagnosis, 398
    diarrhea in, 395
    floctitation in, 394
    in pregnancy, 390
    intestinal hemorrhage in, 396
    lymph-follicles in, 398
    media of infection, 388, 389
    mesenteric glands in, 399
    myocardium in, 395
    perforative peritonitis in, 396
    post-mortem conditions, 400
    respiratory organs in, 397
    roseolæ of, 392
    sequelæ, 397
    spleen in, 393, 400
    stool in, 393
    subsultus tendinum in, 394
    symptoms, 390
    temperature-curve, 391
    thrombosis in, 395
    treatment, 400
    Widal reaction for, 388
  triangle, 392
  ulcer, 399
Typhus, famine-, 312
Typhus fever, 311
    crisis of, 313
    diagnosis, 314
    paralysis of heart in, 313
    rash of, 313
    temperature-curve, 312
    treatment, 314
  war-, 312

Ulcer, gouty, 282
  perforating, 123
Umbilicus, eczema of, 172
Ureter, tuberculosis of, 494
Urethritis, acute gonorrheal, in male, 423
      diagnosis, 427
      sterility after, 425
      treatment, 433, 435
  chronic gonorrheal, in male, 428
  gonorrheal, 422
  granular, 432
  ulcerative, 432
Uridrosis, 195, 416
Urinary organs, tuberculosis of, 491
    treatment, 495
Urine, glucose in, 285
  in diabetes insipidus, 297
    mellitus, 287–290
  sugar in, quantitative determination, 289
    tests for, 287
  tubercle bacilli in, 493
Urocystitis, gonorrheal, 426
Urticaria, 164

Vaccination, 342, 348
  syphilis and, 511
Varicella, 338
  syphilitic, 515
Variola, 341. See also *Small-pox.*
Varioloid, 345
Vasomotor articular neuroses, intermittent, 121
  neuroses of extremities, 120
Veins, gummata of, 529
Venereal crown, 515
Vertebral column, syphilis of, 530
Vertigo, 112
  electric, 113
  epileptic, 91
  essential, 113
Viscera in female, gout of, 282
Vitiligo, 204
  syphilitic, 515
Vocal bands, muscles of, paralysis of, 23

Wandering spleen, 236
War-scurvy, 266
War-typhus, 312

Waxy flexibility of muscles, 114
Weil's disease, 420
Welker's blood-sedimenting method, 240
Whip-worm, 221
Whooping-cough, 374. See also *Pertussis*.
Widal reaction, 388
Word-blindness, 34
Word-deafness, 33, 39, 40
Writers' cramp, 98

XERODERMA, pigmented, 209
  simple, 209

ZOONOSES, 541

GENERAL INDEX.

ABASIA, hysterical, ii. 143
Abdominal dropsy, i. 382
  muscles, peripheral paralysis of, i. 504
    spasm of, i. 512
Aboulia in hysteria, ii. 143
Acanthosis nigricans, ii. 202
Acarus folliculorum, ii. 216
  scabiei, ii. 211
Acephalocysts, i. 352
Achorion Schoenleinii, ii. 222
Acidophile cells, ii. 239
Acne, cachectic, ii. 181
  disseminated, ii. 180
  frontal, ii. 181
  hordeolar, ii. 181
  indurated, ii. 181
  mentagra, ii. 182
  necrotic, ii. 181
  punctate, ii. 181
  pustular, ii. 181
  rosacea, ii. 184
  syphilitic, ii. 181, 515
  toxic, ii. 181
  urticate, ii. 181
  varioliform, ii. 181
  vulgaris, ii. 180
Acoria, i. 240
Acrodynia, ii. 169
Acroparesthesia, ii. 120
Actinomycosis, ii. 546
Addison's disease, i. 468
    treatment, i. 471
Adrenal bodies, diseases of, i. 468
Affrictus pericardiacus, i. 54
Ageusia, i. 531
Agonal edema, i. 124
Agraphia, ii. 41
  amnesic, ii. 42
  motor, ii. 42
  sensory, ii. 42
Akinesis algera, ii. 143, 147
Akromegaly, ii. 136
  partial, ii. 138
Albinism, ii. 204
Albumin in urine, determination, i. 390
Albuminometer, Esbach's, i. 390
Albuminuria, i. 387
  accidental, i. 388
  causes, i. 391
  false, i. 388
  hematogenous, i. 388
  in pneumonia, i. 136
  nephrogenous, i. 388
  pathologic, i. 388
Albuminuria, persistent, i. 388
  physiologic, i. 387
  renal, i. 388
  tests for, i. 388
    Boedecker's, i. 389
    boiling-nitric-acid, i. 388
    Galippe's, i. 389
    Heller's, i. 389
    Panum's, i. 389
  transitory, i. 388
  treatment, i. 392
Albumoses in urine, i. 389
Alcoholic paralysis, i. 539
Alexia, ii. 41
Alkaptonuria, ii. 299
Alopecia, ii. 205
  celsi, ii. 206
  circumscribed, ii. 206
  syphilitic, ii. 515
Amaurosis, uremic, i. 397
American disease, ii. 145
Amimia, ii. 41
  amnesic, ii. 42
  motor, ii. 42
  sensory, ii. 42
Amœba coli, i. 292; ii. 403
Anachlorhydria, i. 240
Anacidity, i. 240
Anadenia, gastric, i. 214
Anal tenesmus, ii. 405
Anarthria, ii. 19
Anemia, bothriocephalus-, i. 297
  brickmakers', i. 311
  miners', i. 311
  progressive pernicious, ii. 250
      anatomic changes, ii. 255
      blood in, ii. 251
      circulatory symptoms in, ii. 253
      diagnosis, ii. 257
      hemorrhages in, ii. 254
      nature of, ii. 257
      nervous system in, ii. 256
      treatment, ii. 257
  tunnel-, i. 311
Anemic fever, ii. 253
Anesthesia, 525
  cutaneous, i. 525
    infectious, i. 527
    paresthesia with, i. 528
    refrigeratory, i. 527
    toxic, i. 527
    traumatic, i. 527
    treatment, i. 528
    vasomotor, i. 527

Anesthesia dolorose, i. 528
  trigeminal, i. 528
    neuroparalytic ophthalmia with, i. 529
Aneurysm, wall of, i. 66
Angina, i. 184
  circumscribed, i. 187
  complications, i. 187
  diffuse, i. 187
  herpetic, ii. 337
  lacunar, i. 187, 188
  Ludwig's, ii. 460
  parenchymatous, i. 187, 189
  pectoris, i. 63
  phlegmonous, i. 187
  superficial, i. 186, 188
  treatment, i. 189
Angioneurotic edema, intermittent, ii. 121
Anguillula intestinalis, i. 315
  stercoralis, i. 315
Anidrosis, ii. 194
Ankylostomiasis, i. 311
  treatment, i. 314
Ankylostomum duodenale, i. 311
Anosmia, i. 529
Anterolateral remnant, i. 546
Ante-stomach, i. 199
Anthrax, ii. 541
  bacillus of, ii. 541
  carbuncle of, ii. 543
  edema of, ii. 543
  pulmonary, and bronchiectasis, i. 109
  treatment, ii. 544
Antidiphtheric serum, ii. 462, 469
Antitoxin, tetanus, ii. 455
Anus, eczema of, ii. 172
Aorta, aneurysm of, i. 65
    diagnosis, i. 70
    neuralgia in, i. 69
    paralysis from, i. 69
    pressure-phenomena, i. 69
    rupture, i. 69
    symptoms, i. 67–70
    treatment, i. 70
    varieties, i. 65
  diseases of, i. 65
  embolism of, i. 72
    gangrene and, i. 72
  isthmus of, constriction and occlusion of, i. 71
Aortic insufficiency with valvular disease, i. 37
  obstruction with valvular disease, i. 38
Ape-hand, i. 496
Aphasia, ii. 39
  amnesic, ii. 39, 40
  ataxic, ii. 39, 44
  cerebral hemiplegia and, ii. 46
  conduction-, ii. 44, 45
  congenital, ii. 47
  cortical, ii. 43
  diagram, ii. 44
  doctrine of, ii. 43
  mixed, ii. 40
  motor, ii. 39
Aphasia, motor, cortical, ii. 44
  optic, ii. 40
  sensory, ii. 33, 39, 40
  simple, ii. 40
  spastic, i. 94
  subcortical, ii. 44
    motor, ii. 45
    sensory, ii. 44
  transcortical, ii. 44
    motor, ii. 44
    sensory, ii. 44
  treatment, ii. 47
Apoplectic attack, ii. 55
    restoration of consciousness after, ii. 56
  habit, ii. 52
Appendicitis, i. 257
  complications, i. 261
  diagnosis, i. 263
  symptoms, i. 260
  treatment, i. 264
Apraxia, ii. 43
Arachnitis, i. 603
Arachnoid hemorrhage, i. 608; ii. 84
Arm and brachial plexus, nerves of, paralysis of, i. 498
  external rotators of, paralysis of, i. 504
  internal rotators of, paralysis of, 503
Arsenical paralysis, i. 538
Arteries, gummata of, ii. 529
Arthritic tophi, ii. 281
Arthritis deformans, ii. 308
  gonorrheal, ii. 426
  of poor, ii. 308
Arytenoid muscles, paralysis of, i. 90
Ascaris lumbricoides, i. 301
  mystax, i. 303
Ascites, i. 382
  cachectic, i. 382
  chylous, i. 383
  fatty, i. 383
  hypostatic, i. 382
  treatment, i. 385
Asiatic cholera, ii. 409
    abdomen in, ii. 415, 417
    anomalies, ii. 415
    bacillus of, ii. 409
    diagnosis, ii. 416
    epidemics of, ii. 412
    fencing attitudes in, ii. 417
    intestines in, ii. 417
    kidneys in, ii. 418
    media of infection, ii. 409–412
    spleen in, ii. 417
    stool of, ii. 413
    symptoms, ii. 413
    treatment, ii. 418
    vomitus of, ii. 414
Aspermatism, i. 464
Asses' cough, ii. 375
Association-fibers, ii. 34
Astasia, hysterical, ii. 143
Asthma, cardiac, i. 30
  dyspeptic, i. 215, 236
  hay-, i. 78
Asthma-cigarets, i. 117
Asthma-crystals, i. 115

Asthma-spirals, i. 115, 116
Ataxia, Friedreich's, i. 592
  hereditary, i. 592
    spinal cord in, i. 595
  locomotor, i. 572. See also *Tabes dorsalis.*
  origin of, i. 577
Athetosis, ii. 106
Atrichia, ii. 205
Atrophy, muscular, infantile, ii. 157
    juvenile, ii. 155
    neural progressive, ii. 158
    progressive myopathic, ii. 153
  spinal progressive muscular, i. 588
      syringomyelia and, i. 592
      treatment, i. 592
Aura, epileptic, ii. 88
Auricle, muscles of, paralysis of, i. 482
Axillary nerve, paralysis of, i. 498
Azoöspermia, i. 464, 465

BACILLI of diphtheria and fibrinous rhinitis, i. 77
Bacillus, comma, ii. 409
  Löffler, ii. 456, 470
  mallei, ii. 545
  of anthrax, ii. 541
  of diphtheria, ii. 456, 470
  of influenza, ii. 380
  of leprosy, ii. 537, 538
  of tetanus, ii. 449
  plague, ii. 372
  tubercle. See *Tubercle bacillus.*
  typhosus, ii. 388
Back, atrophy and weakness of, ii. 155
  muscles of, peripheral paralysis of, i. 504
Bacteria of bronchopneumonia, i. 125, 126
Bacteriuria, i. 457
Balantidium s. Paramœcium coli, i. 293
Balsamics in bronchial catarrh, i. 103
Banting's treatment of obesity, ii. 278
Barlow's disease, ii. 269
Bartholinitis, gonorrheal, ii. 430
Basophile cells, ii. 239
Beard, eczema of, ii. 172
  herpes tonsurans of, ii. 227
Bedbug, ii. 220
Bergeron's electric chorea, ii. 107
Biernier's change in pitch, i. 165,
Biliary calculi, i. 362
  passages, carcinoma of, i. 361
    catarrh of, i. 357
    diseases of, i. 357
    echinococci in, i. 352
    parasites in, i. 361
    purulent inflammation of, i. 359
  sand, i. 362
Black death, ii. 371
Bladder, detrusor of, paralysis of, i. 462
  sphincter of, paralysis of, i, 462
  tuberculosis of, ii. 491
    treatment, ii. 495
  urinary, abscess in, i. 450
    carcinoma, i. 455
    desquamative catarrh of, i. 451
Bladder, urinary, diseases of, i. 448
    foreign bodies in, i. 457
    hyperesthesia of, i. 460
    hypertrophy of, i. 450
      treatment, i. 455
    inflammation of, i. 448. See also *Urocystitis.*
    neuroses of, i. 458
    paralysis of, i. 461
    parasites of, i. 457
    purulent catarrh of, i. 451
    spasm of, i. 460
Bleeders' disease, ii. 270
Blepharospasm, i. 508
Blindness, mind-, ii. 34
  word-, ii. 34
Blood, diseases of, ii. 238
  dry preparations of, ii. 239
  in urine, i. 392
  number of colorless corpuscles in, ii. 240
Blood-sedimenting method, Walker's, ii. 240
Blood-serum reaction of Widal, ii. 388
Blood-shadows, i. 393
Blood-sweating, ii. 142, 195
Blue cough, ii. 376
Boedecker's test for albuminuria, i. 389
Boiling in chest, i. 124
Bone-marrow, involvement of, in leukemia, ii. 243
  lymphoid, ii. 245
  pyoid, ii. 245
  red, ii. 245
Bones, rachitis of, ii. 300
  tertiary syphilis of, ii. 523
Bostock's catarrh, i. 77
Bothriocephalus latus, i. 293, 297
Bothriocephalus-anemia, i. 297
Bowel, intussusception of, i. 271
    nomenclature, i. 272
    treatment, i. 274
  obstruction of, i. 275
    diagnosis, i. 278
    effect on general condition, i. 280
    symptoms, i. 278
    treatment, i. 283
  stenosis of, i. 275
    diagnosis, i. 278
    symptoms, i. 278
    treatment, i. 283
Box-note, i. 114
Brachial plexus and arm, paralysis of nerves of, i. 498
Bracht-Romberg's symptom, i. 539, 576
Bradycardia, paroxysmal, i. 63
  with fat heart, i. 30
Brain, abscess of, ii. 63
    encapsulated, ii. 64
    free, ii. 64
    from otitis interna, ii. 64
    latent, ii. 65
    treatment, ii. 66
  anemia of, ii. 47
  arteries at base of, distribution, ii. 27
  atrophy of, ii. 78

Brain, base of, disease of, focal symptoms in, ii. 39
  convulsions of convexity of, ii. 29
  diffuse sclerosis of, ii. 78
  diseases of, ii. 29
    general symptoms in, ii. 30
    hiccough and, i. 512
    local symptoms in, ii. 30
  edema of, ii. 52
  hemiatrophy of, infantile, ii. 78
  hemorrhage of, 52. See also *Encephalorrhagia.*
  hyperemia of, ii. 50
  hypertrophy of, ii. 77
  inflammation of, ii. 63. See also *Encephalitis.*
  necrotic softening of, ii. 60
  parasites of, ii. 72
  sinuses of, inflammation of, ii. 79
      treatment, ii. 82
    thrombosis of, ii. 79
      treatment, ii. 82
  syphilis of, ii. 530
  tumor of, ii. 68
    latent, ii. 69
    papillitis and, ii. 70
Breast, eczema of, ii. 172
Brickmakers' anemia, i. 311
Bromidrosis, ii. 195
Bronchi, diseases of, i. 96
  tertiary syphilis of, ii. 525
Bronchial asthma, i. 113
    treatment, i. 116
  catarrh, i. 96
    bacteria and, i. 97
    dry, i. 98
    moist, i. 98
    râles in, i. 98
    symptoms, i. 98-101
    treatment, i. 102
  coagulum, i. 104
  constriction, i. 111
  croup, i. 103
  dilatation, i. 106
    treatment, i. 110
  ulcers, i. 97
Bronchiectasis, i. 106
  complications, i. 109
  diagnosis, i. 108
  symptoms, i. 108
  treatment, i. 110
Bronchiolitis, i. 100
  and alveolar emphysema of lungs, i. 118
Bronchitis, fibrinous, i. 103
    treatment, i. 106
  putrid, i. 100
Bronchoblennorrhea, i. 100
Bronchopneumonia, i. 125
Bronchorrhea, i. 100
Bronchostenosis, i. 111
Bronze-disease, i. 469
Bubbling murmurs, i. 166
Bubo, ii. 439
  in syphilis, ii. 513
Bubonic plague, ii. 371
    bacillus of, ii. 372
Bubonic plague, treatment, ii. 374
Bucardia, i. 27
Bulbar monoplegia, ii. 17
  myelitis, acute, ii. 28
  paralysis, acute, ii. 25
    apoplectic, ii. 25
    chronic progressive, ii. 20
      facial expression in, ii. 22
      treatment, ii. 24
    congenital, ii. 24
    myasthenic, ii. 24
Bulimia, i. 240
Burdach's column, i. 546
Bursæ, gout of, ii. 281
  tertiary syphilis of, ii. 523

CACHEXIA, malarial, ii. 368
Cacosmia, i. 530
Cadaveric position, i. 91
Calculi, biliary, i. 362. See also *Gall-stones.*
  carbonate-, i. 441, 446
  cystin, i. 442, 446
  indigo-, i. 442, 446
  mulberry-, i. 363, 441, 446
  oxalate-, i. 441, 446
  phosphatic, i. 441, 446
  preputial, ii. 196
  renal, i. 441
    treatment, i. 446
  uratic, i. 441, 446
  xanthin-, i. 442, 446
Calves, pseudohypertrophy of muscles of, ii. 154
Capillary hemorrhage, ii. 270
Caput Medusæ, i. 335
Carbon-dioxid narcosis, i. 127
Carbuncle, anthrax-, ii. 543
Carcinosis, miliary, i. 150
Cardia, carcinomata of, i. 224
  spasm of, i. 236
Cardiac insufficiency, i. 17. See also *Myocardium, weakness of.*
  intermittency, i. 63
  neuroses, i. 61
Cardiorhexis, i. 34
Cardiospasm, i. 236
Caries, syphilitic, ii. 524
Cartilages, gout of, ii. 281
Catalepsy, ii. 114
Catarrh, acute gastro-intestinal, in infants, i. 247
      treatment, i. 250
  autumnal, i. 77
  Bostock's, i. 77
  calculus-forming, i. 363
  drunkards', i. 212
Cavernous sinus, thrombosis of, ii. 81
Cavities, ii. 205
Cecum, inflammation of, i. 257 See also *Typhlitis.*
Cell, heart-failure, i. 20
Central neuroses with motor disturbances predominant, ii. 87
    with psychic alterations predominant, ii. 139

Central neuroses, with sensory disturbances predominant, ii. 115
    with trophic disturbances predominant, ii. 120
    with vasomotor disturbances predominant, ii. 120
Centrum semi-ovale, disease of, focal symptoms in, ii. 34
Cercomonas coli, i. 293
  intestinalis, i. 293
Cerebellar peduncles, disease of, ii. 39
  tract, lateral, i. 545
Cerebellum, disease of, ii. 38
Cerebral abscess, ii. 63
    encapsulated, ii. 64
    free, ii. 64
    latent, ii. 65
    treatment, ii. 66
  anemia, ii. 47
  arteries, aneurysm of, ii. 73
    embolism of, i. 43; ii. 60
    thrombosis of, ii. 60
  cortex, disease of, focal symptoms, ii. 30
  dura mater, hematomata of, ii. 83
  edema, ii. 52
  embolism, crossed, ii. 61
  hemisphere, median surface of, ii. 31
  hemorrhage, ii. 52. See also *Encephalorrhagia.*
  hyperemia, ii. 50
  meninges, diseases of, ii. 79
    gummata of, ii. 530
    hemorrhage into, ii. 84
  neoplasms, ii. 68
    latent, ii. 69
    papillitis and, ii. 70
  nerves, multiple paralysis of, i. 490
    paralysis of, syphilis and, ii. 531
  pachymeningitis, internal hemorrhagic, ii. 82
  paralysis, acute, of childhood, ii. 67
  parasites, ii. 72
  peduncle, disease of, focal symptoms in, ii. 36
    foot of, ii. 36
    transverse section through, ii. 36, 37
  sinuses, inflammation of, ii. 79
    thrombosis of, ii. 79
Cerebrasthenia, ii. 145
Cerebrospinal fever, 442. See also *Meningitis.*
  meningitis. See *Meningitis.*
  neuron, ii. 32
  sclerosis, multiple, i. 560
    nystagmus in, i. 562
    treatment, i. 563
Cerebrum, horizontal section through, ii. 35
Cervical muscle, spasm of, i. 511
  sympathetic, irritation of, ii. 86
    paralysis of, ii. 85
Cestodes, i. 293. See also *Tapeworms.*
Chancre, hard, ii. 512
    diagnosis, ii. 517
    treatment, ii. 518
  mixed, ii. 441
Chancre, parchment, ii. 512
  soft, ii. 436
    anomalies, ii. 439
    bubo with, ii. 439
    diagnosis, ii. 440
    of lips, ii. 438
    treatment, ii. 441
Chancroid, ii. 436. See also *Chancre, soft.*
Cheiropompholyx, ii. 176
Chest, boiling in, ii. 124
  skin of, scleroderma of, ii. 126
Cheyne-Stokes breathing in fat heart, i. 30
Chiasm, decussation of optic fibers in, diagram, ii. 34
Chicken-breast, ii. 302
Chicken-pox, ii. 338
  temperature-charts, ii. 339
Chloasma, ii. 199
Chlorosis, ii. 258
  blood in, ii. 259
  digestive organs in, ii. 260
  tropical, i. 311
Choked disc, ii. 70
Cholangitis, catarrhal, i. 357
  purulent, i. 359
Cholecystitis, catarrhal, i. 357
  purulent, i. 359
Choledoch duct, carcinoma of mouth of, 361
Cholelithiasis, i. 362. See also *Gall-stones.*
Cholemia, i. 320, 340
Cholera, algid, ii. 413
  Asiatic, ii. 409. See also *Asiatic cholera.*
  asphyctic, ii. 413
  European, ii. 420
  morbus, ii. 420
  nostras, ii. 420
    s. europæa, i. 243
Cholera-diarrhea, ii. 413
Cholera-typhoid, ii. 416
Cholerine, ii. 413
Chorda penis, ii. 424
Chorditis, tuberous, i. 83
  vocalis hypertrophica inferior, i. 83
Chorea, ii. 100
  autotoxic, ii. 101
  diagnosis, ii. 104
  electric, ii. 107
  gait in, ii. 102
  general, ii. 103
  hereditary, in adults, ii. 105
  Huntington's, ii. 105
  of pregnancy, ii. 101
  partial, ii. 103
  posthemiplegic, ii. 105
  prehemiplegic, ii. 105
  reflex, ii. 101
  senile, ii. 100
  symptomatic, ii. 104
  toxic, ii. 101
  treatment, ii. 104
Chromidrosis, ii. 195
Chvostek's facial phenomenon, ii. 97
Chylopericardium, i. 60
Chylothorax, i. 170

Cicatrix, apoplectic, i. 550
Cimex lectularius, ii. 220
Circulatory organs, diseases of, i. 17
    tertiary syphilis of, ii. 528
Cirrhosis, partial, i. 333
Cirsomphalos, i. 335
Clarke's column, i. 543
Claw-hand, i. 496, 497
Clothing-louse, ii. 219
Coccygodynia, i. 524
Coccyx, neuralgia of, i. 524
Colic, hepatic, i. 362
  intestinal, i. 291
  mucous, i. 255
  renal, i. 444, 445
    treatment, i. 447
Colles' law, ii. 532
Color-analytical blood-examination, ii. 239
Colotyphoid, ii. 399
Column of Burdach, i. 546
  of Clarke, i. 543
  of Goll, i. 546
  of Gowers, i. 546
Coma, diabetic, ii. 292
Comedo, ii. 198
Comma bacillus, ii. 409
Commissural fibers, ii. 34
Compensation, derangement of, venous stasis with, i. 42
Compression-myelitis, i. 569, 598
Compression-paralysis, spinal, i. 567
    general paralysis with, i. 569
    treatment, i. 571
Conjunctiva in small-pox, ii. 345
Connective tissue, induration of, in newborn, ii. 203
    subcutaneous, cysticercus of, ii. 220
Consumption, galloping, ii. 480
  pulmonary, ii. 470. See also *Tuberculosis.*
Contact-sensibility, testing of, i. 526
Continued fever ii. 368
Contraction, fascicular muscular, i. 591
Contraction-formula, normal, reversal of, i. 473
Convulsions, uremic, i. 397
Coprostasis, i. 276
Cor adiposum, i. 29
  bovinum, i. 27
  hirsutum, i. 53
Corona radiata, ii. 34
Corpus callosum, disease of, ii. 39
Corpuscles, colorless, in blood, determination of number of, ii. 240
  red, in leukemia, ii. 241
Coryza, i. 73
  complications, i. 74
  hay-, i. 78
  treatment, i. 76
Cough, croupy, ii. 465
  in bronchial catarrh, i. 99
  laryngeal, i. 95
Cowper's gland, gonorrheal inflammation of, ii. 426
Crab-louse, ii. 218
Cramp, choleraic, ii. 415
Cramp, writers', ii. 98
Cramps, i. 513
Cranium, lesions of, ii. 39
Cretinism, sporadic, ii. 128
Crico-arytenoid muscles, posterior, paralysis of, i. 90
Cricothyroid muscles, paralysis of, i. 92
Croup, bronchial, i. 103
  laryngeal, ii. 464
Crural nerve, peripheral paralysis of, i. 505
Crutch-palsy, i. 492
Cutaneous neurosis, ii. 209
  sand, ii. 198
  sensibility, i. 525
Cutis, atrophy of, ii. 208
  testea, ii. 197
Cyanosis, congenital, i. 46
Cysticercus cellulosæ, i. 293
    in brain, ii. 72
  of subcutaneous connective tissue, ii. 220
Cystinuria, ii. 299
Cystoplegia, i. 461
Cystospasm, i. 460

DAMOISEAN'S curves, i. 155
Deafness, word-, ii. 39, 40
Deferentitis, gonorrheal, ii. 425
Defluvium capillorum, ii. 206
Dermatitides, ii. 164
  bulbous, ii. 176
  erythematous, ii. 164
  papular, ii. 190
  pustular, ii. 179
  squamous, ii. 186
  vesicular, ii. 170
Dermatitis, contusiform, ii. 167
  exfoliative, ii. 178
Dermatomycoses, ii. 221
Dermatoses, parasitic, ii. 211
Dermatozoönoses, ii. 211
Dextrocardia, i. 34
Diabetes insipidus, ii. 296
    and mellitus, ii. 297
    urine in, ii. 297
  mellitus, ii. 285
    and insipidus, ii. 297
    coma in, ii. 292
    complications, ii. 291
    deceptive, ii. 287
    diagnosis, ii. 293
    diet in, ii. 295
    idiopathic, ii. 286
    intermittent, ii. 293
    lungs in, ii. 291
    nervous disorders in, ii. 292
    pathogenesis, ii. 294
    quantitative determination of sugar in urine in, ii. 289
    stomach-dilatation in, ii. 291
    symptomatic, ii. 286
    tests for sugar in urine in, ii. 287
    treatment, ii. 294
    urine in, ii. 287-290
Diabetic coma, ii. 292
Diaphragm, paralysis of, i. 491

Diaphragm, spasm of, i. 511
Diarrhea, cholera-, ii. 413
  dentition-, i. 248
  fatty, i. 255
  nervous, i. 290
  summer, of infants, i. 247
Diarrhœa ablactatorum, i. 248
Diathesis, uric acid, ii. 279. See also *Gout.*
Digestive organs, diseases of, i. 175
    tertiary syphilis of, ii. 526
Digitalis-leaves for cardiac insufficiency, i. 23
Diphtheria, ii. 456
  antitoxin for, ii. 462
  bacilli of, ii. 456, 470
  laryngeal, ii. 464
    diagnosis, ii. 467
    serum for, ii. 469
    suffocation in, ii. 466
    treatment, ii. 469
  nasal, ii. 470
  of nasal mucous membranes, ii. 459
  pharyngeal, ii. 456
    diagnosis, ii. 461
    heart in, ii. 462
    nervous affections and, ii. 460
    paralysis with, ii. 460
    serum for, ii. 462
    treatment, ii. 462
    varieties, ii. 458, 459
  scarlatinal, ii. 328
Diplegia, facial, i. 485
Diplococcus pneumoniæ and lanceolatus, i. 129
Dittrich plugs, i. 101
Dorsal muscle, broad, paralysis of, i. 503
Dropsy, abdominal, i. 382
Drum-stick fingers, i. 46, 110
Drunkard's catarrh, i. 212
Drunkards, morning vomiting of, i. 214
Dubini's electric chorea, ii. 107
Duodenitis, acute catarrhal, i. 244, 245
  chronic catarrhal, i. 255
Duodenum, round ulcer of, i. 266
Dura mater and spinal column, relation of, i. 541
    cerebral, hematomata of, ii. 83
    spinal, inflammation of, i. 601
Dysentery, ii. 403
  adynamic, ii. 406
  amebic, ii. 403
  catarrhal, ii. 404
  diagnosis, ii. 408
  follicular, ii. 404
  liver-abscess in, ii. 407
  metastases of, ii. 407
  necrotic, ii. 404
  peritonitis with, ii. 407
  polyarthritis in, ii. 407
  purulent, ii. 404
  putrid, ii. 406
  septic, ii. 406
  stool of, ii. 406
  treatment, ii. 408
Dyspepsia, nervous, i. 241
Dystopia ventriculi, i. 233

Ear, eczema of, ii. 172
Ebstein's treatment of obesity, ii. 278
Echinococci in brain, ii. 73
Echinococcus-cysts, sterile, i. 352
Echokinesis, ii. 108
Eclampsia, ii. 93
  uremic, i. 397
Ecthyma, ii. 179
  syphilitic, ii. 515
Eczema, ii. 170
  marginate, ii. 227
  papular, ii. 170
  treatment, ii. 174
Edema, angioneurotic, intermittent, ii. 121
Ehrlich's color-analytical blood-examination, ii. 239
Elastic fibers in sputum in tuberculosis, ii. 476
Electric-sense of skin, testing of, i. 526
Empyema pleuræ necessitatis, i. 157
Encephalitis, ii. 63
  congenital, ii. 68
  hemorrhagic, ii. 66
Encephalomalacia, inflammatory, ii. 66
Encephalorrhagia, ii. 52
  amount of blood extravasated in, ii. 54
  dementia in, ii. 58
  diagnosis, ii. 58
  stages, ii. 55
  symptoms, ii. 55–58
  treatment, ii. 59
Endocarditis, i. 47
  chronic, i. 36
  contracting, i. 47
  cryptogenetic, i. 47
  fetal, and heart-disease, i. 46
  recurrent contracting, i. 47
  ulcerative, i. 47
    and puerperal fever, i. 47
    and typhoid fever, i. 49
    treatment, i. 49
  verrucose, i. 47, 49
    and rheumatism, i. 50
    and scarlet fever, i. 50
Endocardium, diseases of, i. 35
  inflammation of, i. 47. See also *Endocarditis.*
English sweating-sickness, ii. 338
Enormitas cordis, i. 27
Enteralgia, nervous, i. 291
Enteric fever, ii. 387. See also *Typhoid fever.*
Enteritis, membranous, i. 255
Enteroptosis, i. 288
Enterorrhagia of newborn, i. 288
Enterostenosis, i. 275. See also *Bowel, stenosis of.*
Enuresis, diurnal, i. 458
  nocturnal, i. 458
Eosinophile cells, ii. 239
Ephelides, ii. 199
Epidemic cerebrospinal meningitis, ii. 442. See also *Meningitis.*
Epidermis, hypertrophy of, ii. 200
Epidermolysis, hereditary, bulbous, ii. 178

Epididymitis, gonorrheal, ii. 425
Epidural hemorrhage, i. 608
Epigastrium, restless movements in, i. 236
Epiglottis, depressor of, paralysis of, ii. 22
Epilepsy, ii. 87
  acute, ii. 93
  cortical, ii. 33, 87
  diagnosis, ii. 92
  diurnal, ii. 90
  grave, ii. 88
  idiopathic, ii. 87
  Jacksonian, ii. 33, 87
  late, ii. 88
  major, ii. 88
  mild, ii. 91
  minor, ii. 91
  nocturnal, ii. 90
  procursive, ii. 92
  reflex, ii. 88
  symptomatic, ii. 87
  toxic, ii. 87
  treatment, ii. 93
  true, ii. 87
Epileptic attack, ii. 89
  aura, ii. 88
  equivalents, ii. 91
Epileptoid states, ii. 91
Epizoötic aphthæ, ii. 548
Erb's paralysis of brachial plexus, i. 499
  supraclavicular point, i. 499
Ergotism, i. 539
Ergot-poisoning, paralysis from, i. 539
Eructation, i. 211
  nervous, i. 235
Erysipelas, ii. 329
  afebrile, ii. 332
  bulbous, ii. 332
  cutaneous, ii. 332
  gangrenous, ii. 333
  of mucous membranes, ii. 333
  pustular, ii. 332
  treatment, ii. 333
  vesicular, ii. 332
  wandering, ii. 333
Erythema, annular, ii. 168
  gyrate, ii. 168
  iris, ii. 168
  mammillatum, ii. 168
  multiform exudative, ii. 168
  nodose, ii. 166
  recurring scarlatiniform, ii. 169
  syphilitic, ii. 514
Erythrasma, ii. 229
Erythromelalgia, ii. 121
Esbach's albuminometer, i. 390
  reagents, i. 389
Esophagism, i. 204
Esophagitis, catarrhal, i. 200
  phlegmonous, i. 201
Esophagomalacia, i. 202
Esophagus, carcinoma of, i. 191
Esophagus, carcinoma of, treatment, i. 196
  catarrh of, i. 200
  dilatation of, i. 199
  dimensions of, i. 194
  diseases of, i. 191
  diverticula of, i. 197
    pulsion-, i. 198
    traction-, i. 198
  mucous membrane of, in small-pox, ii. 345
  muscles of, paralysis of, ii. 23
  paralysis of, i. 203
  peptic ulcer of, i. 201
  softening of, i. 202
  spasm of, i. 204
  spontaneous rupture of, i. 202
  stenosis of, i. 196
    and carcinoma, i. 192, 193
  tertiary syphilis of, ii. 527
  thrush of, i. 203
Esthesiometer, i. 526
État mamelonné, i. 213
European cholera, ii. 420
Exanthemata, acute infectious, ii. 311
  syphilitic, ii. 514
Exophthalmic goiter, ii. 131
  treatment, ii. 134
Exophthalmos, ii. 132
Expectorants, i. 102
Exploratory puncture, apparatus, i. 160
Extremities, arteries of, embolism of, i. 43
  eczema of, ii. 173
Eyebrow, corrugator muscle of, paralysis of, i. 481
Eyelids, orbicular muscle of, paralysis of, i. 481

FACE, eczema of, ii. 171
  muscles of, spasm of, i. 508
  paralysis of muscles of, i. 478
  seborrhea of, ii. 196
Facial nerve, diagram of distribution, i. 487
    paralysis of, i. 477
      diagnosis, i. 486
      treatment, i. 488
Facies, choleraic, ii. 414
  myopathic, ii. 157
  tetanic, ii. 453
Famine-typhus, ii. 312
Fasciæ, tertiary syphilis of, ii. 523
Fasciculus, anterolateral, i. 546
Favus, ii. 222
  confertus, ii. 225
Favus-bodies, ii. 224
Febris urticata, ii. 164
Fecal concretions, true, i. 259
    false, i. 258
Feet, sweating of, ii. 194
Feline tongue, ii. 326
Fermentation-test for sugar in urine, ii.

Fingers, eczema of, ii. 173
  drum-stick, i. 46, 110
Fistula, bimucous intestinal, i. 281
  pulmonary, i. 166
Fleas, ii. 220
  sand, ii. 220
Flexibility of muscles, waxy, ii. 114
Foot-and-mouth disease, ii. 548
Fortification-scotomata, ii. 119
Four-glass test, i. 210
Freckles, ii. 99
Frémissement cataire, i. 36
Fremitus, bronchial, i. 98
Friedreich's ataxia, i. 592
Fright-paralysis, i. 601

GALIPPE'S picric-acid test, i. 389
Gall-bladder, carcinoma of, i. 361
  dropsy of, i. 360
  empyema of, i. 359
Gall-stones, i. 362
  facetted, i. 363
  treatment, i. 366
Ganglion-cells of anterior horns, disease of, i. 582
    in adults, inflammation of, i. 586
  pigmentary degeneration of, i. 588
Gangrene and embolism of aorta, i. 72
  from perichondritis of larynx, i. 88
  symmetrical, ii. 122
Garrod's thread-test, ii. 282
Gastralgia, nervous, i. 238
Gastrectasis, i. 227
  treatment, i. 231
Gastric catarrh, acute, i. 210
    chronic, i. 212
      treatment, i. 215
    hypertrophic, i. 213
  juice, digestive power of, i. 206
    hydrochloric acid in, i. 207, 209
    hypersecretion of, i. 241
    lab-ferment in, i. 210
    pepsin in, i. 209
Gastritis, chronic atrophic, i. 213
    cystic, i. 214
    polypous, i. 213
  phlegmonous, i. 216
  purulent, i. 216
Gastro-intestinal catarrh, acute, in infants, i. 247
      treatment, i. 250
Gastroptosis, i. 229, 233
Gastrosuccorrhea, i. 241
Gastroxynsis, i. 242
Genitalia, eczema of, ii. 172
  seborrhea of, ii. 196
Genito-urinary organs, diseases of, i. 387
    tertiary syphilis of, ii. 528
Gerlier's disease, ii. 113
Giant-growth, morbid, ii. 136
  partial, ii. 138
Gingivitis, scorbutic, ii. 266, 267
Glanders, ii. 544
Glans penis, neuralgia of, i. 524
Gliomatosis, i. 565
Globus, hysterical, ii. 140
Glomerulonephritis, i. 404
Glossitis, variolous, ii. 344
Glossopharyngeal nucleus, situation, ii. 20
Glossoplegia, i. 489
Glottis, edema of, i. 85
    disease of larynx and, i. 86
    spontaneous, i. 86
  spasm of, i. 93
    phonatory, i. 94
    rachitis and, i. 94
    tetany and, ii. 97
Gluteal muscles, pseudohypertrophy of, ii. 155
  nerves, peripheral paralysis of, i. 506
Glycosuria, alimentary, ii. 294
  hematogenous, ii. 294
  myogenous, ii. 294
  nephrogenous, ii. 294
  neurogenous, ii. 294
Gmelin's test for biliary coloring-matter, i. 319
Goiter, exophthalmic, ii. 131
    treatment, ii. 134
"Golden vein," i. 286
Goll, column of, i. 546
Gonococci, ii. 421
Gonorrhea, ii. 421
  acute, in female, ii. 430
      treatment, ii. 436
    in male, ii. 423
      treatment, ii. 433
  chronic, in female, ii. 431
      treatment, ii. 436
    in male, ii. 428
      treatment, ii. 435
  diagnosis, ii. 423
  gonococci of, ii. 421
  gouty, ii. 427
  in children, ii. 422
  morning-drop in, ii. 428
  treatment, ii. 433
  two-glass test in, ii. 429
  urethral injections in, ii. 433
Gonorrheal Bartholinitis, ii. 430
  metastases, ii. 424, 426
  threads, ii. 428
Gout, ii. 279
  heart in, ii. 283
  kidneys in, ii. 283
  nature of, ii. 284
  treatment, ii. 284
Gouty deposits, explanation, ii. 284
  nodules, ii. 281
  ulcers, ii. 282
Gowers' column, i. 546
Graefe symptom, ii. 133
Graves' disease, ii. 131
    treatment, ii. 134
Gray matter of spinal cord, i. 541
Grip, ii. 380. See also *Influenza.*
Gummata, ii. 522-532
Günzburg's phloroglucin-vanillin solutions, i. 207
Gustatory nerve, disease of, i. 530

HAFFKINE's prophylaxis, ii. 374
Hagen-Brandt's formula to abort acute coryza, i. 76
Hair, atrophic changes in, ii. 205
  brittleness of, ii. 207
  deficiency of, ii. 205
  graying of, ii. 205
  hypertrophy of, ii. 202
  loss of, ii. 205
Ham-spleen, ii. 234
Hand, ape-, i. 496
  claw-, i. 496, 497
  palm of, eczema of, ii. 173
  sweating of, ii. 194
Hay-asthma, i. 78
Hay-coryza, i. 78
Hay-fever, i. 77
Head, muscles of, spasm of, i. 511
Headache, habitual, ii. 115
  nervous, ii. 115
Head-louse, ii. 216
Heart, acquired valvular disease of, i. 35
      cardiac manifestations in, i. 37
      diagnosis, i. 43
      embolic alterations, i. 42
      spontaneous recovery, i. 44
      symptoms, i. 37-43
      treatment, i. 45
      venous stasis with, i. 42
  beer-, Munich, i. 28
  chronic aneurysm of, after myocarditis, i. 33
  dilatation of, i. 24
    acute, blood-pressure and, i. 25
    and cardiac insufficiency, i. 27
    and pericarditis, i. 57
    diminished resistance of heart muscle and, i. 24
    treatment, i. 28
  echinococcus of, i. 34
  fat, i. 29
    anemic, i. 30
    cachectic, i. 30
    in obesity, ii, 277
    plethora and, i. 30
  hypertrophy of, i. 28
    toxic, i. 29
  new-growths of, i. 33
  ox-, i. 27
  right-sided, i. 34
  rupture of, i. 34
  thrombosis of, i. 51
  villous, i. 52
Heartburn, i. 211
Heart-disease, congenital, i. 45
Heart-failure cells, i. 20
Heart-murmurs, localization, i. 43
Heart-pain, nervous, i. 63
Heberden's nodes, ii. 308
Hectic fever, ii. 480
  flush, ii. 480
Hematomyelitis, i. 548, 553
Hematoporphyrinuria, i. 396
Hematuria, i. 392
  Heller's test, i. 393
  winter, ii. 272
Hemianesthesia, hysteric, ii. 141
Hemianopsia, ii. 33
Hemiatrophy, progressive facial, ii. 123
Hemichorea, ii. 103
Hemicrania, ii. 117
  alternating, ii. 118
  ophthalmic, ii. 119
  sympathetic-paralytic, ii. 119
  sympathetic-spastic, ii. 119
  treatment, ii. 120
  vasomotor, ii. 119
Hemicranic equivalent, ii. 120
Hemidrosis, ii. 142, 195
Hemihypertrophy, facial, ii. 125
Hemiplegia, alternating, ii. 25
  cerebral, ii. 36
  cortical, development, ii. 32
  crossed, ii. 17
  facial, i. 485
Hemoglobinemia, ii. 272
Hemoglobinuria, i. 392, 394
  paroxysmal, ii. 272
Hemopericardium, i. 60
Hemophilia, ii. 270
Hemopneumopericardium, i. 58
Hemopneumothorax, i. 162
Hemoptysis and fibrinous bronchitis, i. 105
Hemorrhoidal nodules, inflammation of, i. 286
Hemorrhoids, i. 284
  blind, i. 286
  mucous, i. 286
  treatment, i. 287
Hemothorax, i. 170
Hepatic artery, aneurysm of, i. 370
    embolism of, i. 43
Hepatitis, chronic interstitial, i. 331. See also *Liver, cirrhosis of.*
  suppurative, i. 327
    treatment, i. 330
Hepatoptosis, i. 355
Herpes, progenital, ii. 337
  tonsurans, ii. 226
  with pneumonia, i. 137
  zoster, ii. 334
    neuralgia with, ii. 335
Hiccough, i. 511
Hirsuties, ii. 202
Hives, ii. 164
Horns, anterior, ganglion-cells of, disease of, i. 582
    in adults, inflammation of ganglion-cells of, i. 586
Hunger, disorders of sense of, i. 240
Huntington's chorea, ii. 105

Hydrocephalus, circumscribed, ii. 76
  congenital, ii. 74
  external, ii. 76
  intermeningeal, ii. 76
  internal, ii. 76
  ventricular, ii. 76
Hydromyelia, i. 564
Hydronephrosis, i. 435
  and pyelitis, i. 440
  partial, i. 436
  treatment, i. 438
  varieties, i. 437
Hydropericardium, i. 59
Hydrophobia, ii. 549
Hydropneumopericardium, i. 58
Hydropneumothorax, i. 162
  diagnosis, i. 166
  encapsulated, i. 162, 167
  subphrenic, i. 167
  treatment, i. 168
Hydrothorax, i. 169
Hypacidity, i. 208, 240
Hyperacidity, i. 208, 209, 240
Hyperacusis, Willisian, i. 482
Hyperchlorhydria, i. 240
Hypergeusia, i. 531
Hyperidrosis, ii. 193
Hyperkinesis, i. 507
Hyperosmia, i. 529
Hyperthyroidism, ii. 134
Hypertrophy, cardiac, eccentric, i. 24
  muscular, true, ii. 159
    with juvenile atrophy, ii. 156
  pseudomuscular, ii. 153, 158
Hypochlorhydria, i. 240
Hypogeusia, i. 531
Hypoglossal nucleus, situation, ii. 20
  nerve, paralysis of, i. 489
  spasm, i. 510
Hyposmia, i. 529
Hysteria, ii. 139
  diagnosis, ii. 144
  eye in, ii. 142
  hearing in, ii. 142
  paralysis with, ii. 140
  treatment, ii. 144
Hysterical globus, ii. 140
Hystero-epilepsy, ii. 143

ICHTHYOSIS, ii. 200
  sebaceous, ii. 197
Icterus, i. 315. See also *Jaundice.*
  viridis, i. 324
    in cardiac insufficiency, i. 20
Ileocolitis, i. 244
Ileus, i. 275. See also *Bowel, obstruction of.*
  paralytic, i. 277
Iliopsoas muscle, paralysis of, i. 505
Impetigo, ii. 179
  syphilitic, ii. 515
Impotence in male, i. 463
Infants, acute gastro-intestinal catarrh in, i. 247
      treatment, i. 250
  fatty liver in, i. 342
  feeding of, with milk substitutes, i. 252
Infectious diseases, ii. 311
    of typical localization, ii. 311
    of varying localization, ii. 456
    with changes in blood, ii. 357
      in blood-generating organs, ii. 357
      in digestive organs, ii. 384
      in joints and muscles, ii. 349
      in nervous system, ii. 442
      in respiratory organs, ii. 374
    with lesions of sexual organs, ii. 421
Inferior oblique muscle of head, spasm of, i. 511
Influenza, ii. 380
  abdominal, ii. 383
  bacillus of, ii. 380
  pneumonia with, ii. 382
  treatment, ii. 383
Insanity, pre-epileptic, ii. 89
Intention-tremor, i. 561
Intermittent fever, ii. 364
    comatose, ii. 367
    duplicated, ii. 367
    larval, ii. 367.
    pernicious, ii. 367
    quotidian, ii. 364-366
    tertian, ii. 366
    treatment, ii. 371
Internal capsule, disease of, focal symptoms in, ii. 36
Intestinal crises, i. 291
Intestine, animal parasites of, i. 292
  atony of, i. 289
  carcinoma of, i. 267
    treatment, i. 270
  catarrh of, acute, i. 242
      symptoms and diagnosis, i. 244
      treatment, i. 246
    chronic, i. 252
      treatment, i. 256
  diseases of, i. 242
  hemorrhage of, in newborn, i. 288
  nervous spasm of, i. 290
  neuroses of, i. 289
    motor, i. 289
    sensory, i. 290
  peristaltic unrest of, i. 290
  polypi of, i. 270
  protozoa in, i. 292
  relaxation of muscular coat of, i. 289
  sarcoma of, i. 270
  tertiary syphilis of, ii. 527
  tuberculosis of, ii. 488
  tuberculous ulcers of, ii. 490
  worms of, i. 293
Intussusception, i. 271
Intussusceptum, i. 271
Intussuscipiens, i. 271
Invagination, i. 271
Isthmus aortæ persistens, i. 71
  of aorta, constriction and occlusion of, i. 71
Itch, ii. 211
  treatment, ii. 215
Itch-burrow, ii. 214
Itch-parasite, ii. 212
Itching-sense, testing of, i. 526
Itching-weeping tetter, ii. 170

JACKSONIAN epilepsy, ii. 33, 87
Jaffe's test for indican in urine, i. 281
Jaundice, i. 315
  catarrhal, i. 358
  cutaneous, i. 318
  diagnosis, i. 321
  diffusive, i. 316
  due to pleiochromia, i. 316
  due to polycholia, i. 316
  gastroduodenal, i. 317, 358
  hepatogenous, i. 316
  hypertrophic cirrhosis of liver with, i. 337
  menstrual, i. 357
  of mucous membranes, i. 318
  toxic, i. 317
  treatment, i. 322
Jejunitis, i. 244, 246
Joints, dropsy of, intermittent, ii. 121
  infectious diseases with changes in, ii. 349
  tertiary syphilis of, ii. 524

KEEL-BREAST, ii. 302
Keratosis, ii. 200
Kidneys, absence of, i. 432
  adenoma of, i. 425
  amyloid, i. 421
  arteriosclerotic contracted, i. 411, 421
  atrophy of, granular, i. 411
  carcinoma of, i. 423
  contracted, i. 411
    cyanotic, i. 401
    diagnosis, i. 413
    hypertrophy of myocardium and, i. 414
    pulse-tracing of, i. 415
    symptoms, i. 413
    treatment, i. 416
  cyanosis of, i. 22
  cyanotic induration of, i. 23, 401
  cystadenoma of, i. 426
  cystic, i. 425
  diseases of, i. 387
  dystopia of, i. 432
  echinococcus of, i. 427
  embolic infarction of, i. 420
  horseshoe, i. 432
  hypostatic, i. 401
    in cardiac insufficiency, i. 19
  large white, i. 409
  movable, i. 428
    treatment, i. 431
  pelvis of, calculi in, i. 441
      treatment, i. 446
    carcinoma of, i. 447
    dilatation of, i. 435
      treatment, i. 438
      varieties, i. 437
    diseases of, i. 435
    inflammation of, i. 438
    parasites of, i. 448
    tuberculosis of, ii. 492
  sarcoma of, i. 425
  spotted contracted, i. 413
  suppuration of, i. 416
    treatment, i. 420
Kidneys, surgical, i. 417
  tuberculosis of, ii. 491
    treatment, ii. 495
  venous hyperemia of, i. 401
  wandering, i. 264, 428
    incarceration of, i. 430
    treatment, i. 431

LABIA majora, neuralgia of, i. 524
Laennec's cirrhosis of liver, 333
Lagophthalmos, choleraic, ii. 414
  paralytic, i. 481
Laryngeal cough, i. 95
  mucous membrane, anesthesia of, i. 95
      in small-pox, ii. 345
      sensory disorders of, i. 95
Laryngitis, catarrhal, i. 79
    treatment, i. 83
  granular, i. 82
  hemorrhagic, i. 81
  herpetic, ii. 337
Larynx, abscess of, i. 85
  catarrh of, i. 79
    treatment, i. 83
  consumption of, ii. 486
  diseases of, i. 79
    edema of glottis and, i. 86
  hyperesthesia of, i. 95
  muscles of, paralysis of, i. 89
      treatment, i. 92
  mycosis of, i. 96
  pachydermia of, i. 82
  papillomata of, i. 83, 84
  paresthesia of, i. 95
  perichondritis of, i. 87
  phthisis of, ii. 486
  polypi of, i. 83, 84
  tertiary syphilis of, ii. 525
  ulcers of, treatment, i. 84
Lead-paralysis, i. 536
Legs, eczema of, ii. 173
Lentigenes, ii. 199
Leontiasis ossea, ii. 138
Leprosy, ii. 537
  bacillus of, ii. 537, 538
  cutaneous, ii. 539
  diagnosis, ii. 540
  facies in, ii. 539
  nervous, ii. 540
  treatment, ii. 541
  ulcers of, ii. 539
Leprous leontiasis, ii. 539
  satyriasis, ii. 539
Leptothrices, pulmonary, i. 101
Leukemia, ii. 238
  blood in, ii. 239
  colorless corpuscles in, ii. 239
  complications, ii. 244
  diagnosis, ii. 245
  involvement of bone-marrow in, ii. 243
  lymphatic, ii. 243
  nature of, ii. 246
  onset, ii. 243
  red corpuscles in, ii. 241
  spleen in, ii. 242
  splenic enlargement in, ii. 242
  treatment, ii, 246

Leukoderma, ii. 204
  syphilitic, ii. 515
Leukomyelitis, i. 554
  chronic, i. 558
Leukoplakia oris, i. 181
Lice, ii. 216
  clothing-, ii. 219
  crab-, ii. 218
  head-, ii. 216
Lichen of scrofulous, ii. 191
  ruber, ii. 191
    planus, ii. 192
  syphilitic, ii. 515
Lientery, i. 245, 254
Lips, eczema of, ii. 172
Little's disease, i. 581
Liver, abscess of, i. 327
    treatment, i. 330
  adenoma of, i. 350
  adipose, i. 342
  amyloid, i. 343
  atrophy of, acute yellow, i. 338
      treatment, i. 341
    cryptogenetic, i. 339
    granular, i. 332
  blood-vessels, diseases of, i. 368
  carcinoma of, i. 345
    treatment, i. 350
  cast of, i. 325
  cirrhosis of, i. 331
    alcoholic, i. 332, 334-336
    arteriosclerotic, i. 334, 338
    atrophic, i. 332
    diagnosis, i. 334
    hypertrophic, i. 332
      biliary, i. 333, 337
    Laennec's, i. 333
    monocellular, i. 334
    monolobular, i. 333
    multilobular, i. 333
    senile, i. 334, 338
    symptoms, i. 334
    syphilitic, i. 334, 337
    treatment, i. 338
  constricted, i. 355
  contracted, i. 331. See also *Liver, cirrhosis of.*
  diseases of, i. 315
  displacements of, i. 355
  echinococcus of, i. 350
    treatment, i. 355
  fatty, i. 342
    in obesity, ii. 277
    infiltration of, i. 342
  fissured, i. 355
  hypostatic, i. 323
    in cardiac insufficiency, i. 20
    induration of, cyanotic, i. 23
  mold of, i. 325
  movable, i. 355
  nutmeg-, i. 323, 324
    cyanotic, i. 23
  sarcoma of, i. 350
  serous coat of, inflammation of, i. 325
  suppurative inflammation of, i. 327
      treatment, i. 330
  syphilitic lobulated, i. 334; ii. 528
Liver, tertiary syphilis of, ii. 527
  venous hyperemia of, i. 323
  wandering, i. 355
Localization, sense of, i. 526
Lockjaw, ii. 452
Locomotor ataxia, i. 572. See also *Tabes dorsalis.*
Löffler's bacillus, ii. 456, 470
Lower extremities, muscular spasm in, i. 513
Ludwig's angina, ii. 460
Lues, ii. 510. See also *Syphilis.*
Lumbago, ii. 356
Lung, abscess-formation of, bronchiectasis and, i. 108
  atelectasis of, i. 121
  brown induration of, i. 22
  carcinoma of, i. 149
  catarrhal inflammation of, i. 125
    treatment, i. 128
  cirrhosis of, i. 139
    treatment, i. 142
  diseases of, i. 117
  echinococcus of, i. 151
  edema of, i. 123
    agonal, i. 124
  emphysema of, alveolar, i. 117
      bronchiolitis and, i. 118
      complications, i. 119
      treatment, i. 120
    interstitial, i. 120
    vicarious alveolar, i. 119
  fibrinous inflammation of, i. 128. See also *Pneumonia, fibrinous.*
  gangrene of, i. 144
    complications, i. 147
    treatment, i. 148
  hypostasis of, i. 122
  new-growth of, i. 149
  sarcoma of, i. 149
  slaty induration of, i. 140
  suppuration of, i. 142
  tertiary syphilis of, ii. 526
  tuberculosis of, ii. 470. See also *Tuberculosis, chronic pulmonary.*
  tumors of, i. 149
Lymphatic glands, scrofulous enlargement of, ii. 506
Lyssa, ii. 519

MALARIAL cachexia, ii. 368
  fever, ii. 361
    diagnosis, ii. 364
    melanemia and, ii. 368
    mosquitoes and, ii. 364
    plasmodia of, ii. 362
    symptoms, ii. 364
    treatment, ii. 369
Male, impotence in, i. 463
  sexual organs, diseases of, i. 463
  sterility in, i. 464
Maliaria, ii. 175
Mammary gland, gummata of, ii. 526
    neuralgia of, i. 521
Mange, ii. 212
Maréchal's test for biliary coloring-matter, i. 319

- Mast-cells, ii. 240
- Mastication, muscles of, paralysis of, i. 476
  - spasm of muscles of, i. 507
- Masticatory spasm, i. 507
- Mastodynia, i. 521
- Measles, ii. 316
  - anomalies of, ii. 319
  - diagnosis, ii. 320
  - eruption of, ii. 318
  - laryngeal complications, ii. 319
  - otitis media in, ii. 320
  - sequelæ, ii. 320
  - temperature-curve, ii. 317
  - treatment, ii. 321
- Measles-pneumonia, ii. 320
- Median nerve, paralysis of, i. 495
- Mediastinitis, i. 173
- Mediastinopericarditis, i. 53
- Mediastinum, abscess of, i. 174
  - diseases of, i. 171
  - inflammation of, i. 173
  - interstitial emphysema of, i. 174
  - tumors of, i. 171
- Medulla oblongata, acute inflammation of, ii. 28
  - arteries of, embolism of, ii. 26
    - thrombosis of, ii. 26
  - diseases of, ii. 17
  - hemorrhage into, ii. 25
  - neoplasms of, ii. 28
  - transverse section through, ii. 21
- Megaloblasts, ii. 252
- Megalogastria, i. 229
- Megastomum entericum, i. 293
- Melanemia, ii. 368
- Melanoderma, syphilitic, ii. 515
- Melanosis, villous, i. 253
- Melasicterus, i. 318
- Melena of newborn, i. 288
- Ménière's disease, ii. 113
- Meningeal hemorrhage, i. 607
- Meninges, cerebral, diseases of, ii. 79
    - gummata of, ii. 530
    - hemorrhage into, ii. 84
  - spinal, diseases of, i. 601
    - neoplasms of, i. 609
- Meningitic spots, ii. 444
- Meningitis, abortive, ii. 445
  - apoplectiform, ii. 445
  - cerebrospinal, i. 604
    - epidemic, ii. 442
      - anatomic changes, ii. 446
      - diagnosis, ii. 446
      - treatment, ii. 447
    - purulent, in pneumonia, i. 136
    - secondary purulent, ii. 448
    - serous, ii. 449
    - tuberculous, ii. 498
      - abdomen in, ii. 501
      - diagnosis, ii. 502
      - respiration in, ii. 501
      - spinal cord in, ii. 499
      - symptoms, ii. 500
      - treatment, ii. 502
  - fulminating, ii. 445
  - intermittent, ii. 445
- Meningitis, spinal, i. 603
  - acute, i. 603
  - chronic, i. 606
- Meningococcus intracellularis, ii. 442
- Meningotyphoid, ii. 394
- Mercurialism, ii. 520
- Merycism, i. 237
- Mesenteric artery, embolism of, i. 43
- Metabolism, disorders of, ii. 275
- Metastases, gonorrheal, ii. 424, 426
- Methylene-blue for malaria, ii. 370
- Miasm, ii. 362
- Microsporon furfur, ii. 221
- Migrain, ii. 117. See also *Hemicrania.*
- Migrainous status, ii. 118
- Miliary fever, ii. 338
  - tuberculosis, ii. 495
- Milium, ii. 198
- Mind-blindness, ii. 34
- Miners' anemia, i. 311
- Miserere, i. 273, 280
- Mitral insufficiency with valvular disease, i. 39
  - obstruction with valvular disease, i. 40
  - stenosis with valvular disease, i. 40
- Mogigraphia, ii. 98
- Moist tetter, ii. 170
- Molimina, hemorrhoidal, i. 286
- Monophasia, ii. 40
- Monoplegia, ii. 32
  - bulbar, ii. 17
- Moore's test for sugar in urine, ii. 287
- Morbilli, ii. 316. See also *Measles.*
- Morbus cœruleus, i. 46
  - maculosus Werlhofii, ii. 264
- Morning-drop, ii. 428
- Morvan's disease, i. 567
- Mosquitoes and malaria, ii. 364
- Mother-mark, ii. 199
- Motor cortical centers, ii. 30, 31
  - nerves, spasmodic disorders of, i. 507
    - inflammation of, i. 533
  - trigeminal spasm, i. 507
- Mouth, diseases of, i. 175
  - inflammation of, i. 175 *et seq.*
  - thrush of, i. 179
- Movable spleen, ii. 236
- Mulberry-calculi, i. 363, 441, 446
- Mumps, ii. 384
  - orchitis in, ii. 386
- Munich beer-heart, i. 28
- Murmur, bubbling, i. 166
  - endocardial and pericardial, i. 56
  - nun's, i. 313
  - presystolic, i. 40
- Muscles, diseases of, ii. 153
  - infectious diseases with changes in, ii. 349
  - tertiary syphilis of, ii. 523
- Muscular contraction, fascicular, i. 591
  - spasm, i. 507
- Musculocutaneous nerve, paralysis of, i. 498
- Myasthenia, ii. 163
- Mycosis nasi, i. 79
  - pharyngis leptothricia, i. 190

Mycotic bronchial plugs, i. 101
Myelitis, acute, i. 552
    anesthesia with, i. 554
    ascending, i. 555
    bed-sores with, i. 556
    diagnosis, i. 556
    gangrene of skin in, i. 555
    treatment, i. 557
  bulbar, acute, ii. 28
  central, i. 554
  chronic, i. 558
    ascending, i. 559
    central, i. 558
  circumscribed, i. 554
  compression-, i. 569, 598
  diffuse, i. 554
  hemorrhagic, i. 553
  multiple, i. 554
  purulent, i. 553
  transverse, i. 554
Myelocytes, ii. 240
Myelomalacia, inflammatory, i. 553
  necrotic, i. 553
Myelomeningitis, acute, i. 554
  chronic, i. 558
Myocarditic cicatrices, i. 31
Myocarditis, i. 31
  chronic, i. 32
    aneurysm after, i. 33
  treatment, i. 33
  varieties, i. 32
Myocardium, diseases of, i. 17
    weakness and, i. 21
  echinococcus of, i. 34
  hyperirritability of, after pericarditis, i. 56
  hypertrophy of, and contracted kidney, i. 411
  inflammation of, i. 31
  tertiary syphilis of, ii. 528
  tumors of, i. 33
  weakness of, i. 17
    and dilatation of heart, i. 27
    diseases and, i. 21
    in pericarditis, i. 55
    senile, i. 22
    toxic varieties, i. 22
    treatment, i. 23
    venous stasis with, i. 42
Myoclonus, ii. 107
Myomalacia cordis, i. 31
Myosis, uremic, i. 397
Myositis from trichinæ, i. 307
  progressive ossifying, ii. 160
Myotonia, ii. 162
Myxedema, ii. 128, 138
Myxœdema congenitum, ii. 130
  strumiprivum, ii. 129

NAILS, atrophic disorders of, ii. 208
  favus of, ii. 225
  hypertrophy of, ii. 203
Narco-epilepsy, ii. 92
Narcosis, carbon-dioxid, i. 127
Nasal mucous membrane, catarrh of, from coryza, i. 75
    fibrinous inflammation of, i. 77
    fungi on, i. 79
    molds on, i. 79
Neck, inferior subcutaneous nerve of, neuralgia of, i. 519
Nemathelminthes, i. 301
Neoplasmata pericardii, i. 60
Nephritis, acute desquamative, i. 404
    lymphomatous, i. 404
  chronic hemorrhagic, i. 410
    interstitial, i. 411
      diagnosis, i. 413
      pulse-tracing of, i. 415
      symptoms, i. 413
      treatment, i. 416
    parenchymatous, i. 409
  diffuse, i. 402
    acute, i. 402
      pulse-tracings, i. 406
      treatment, i. 408
      urinary sediments from, i. 405
  purulent, i. 416
    cryptogenetic, i. 417
    treatment, i. 420
  scarlatinal, ii. 328
Nephrolithiasis, i. 441
  diagnosis, i. 443
  symptoms, i. 443
  treatment, i. 446
Nerve, electric irritability of, i. 474
Nerve-pain, i. 513
Nerve-roots, spinal, changes in, in tabes dorsalis, i. 574
    paralysis of, i. 602
Nervous disorders, functional, ii. 86
  fever, ii. 394
  system, diseases of, i. 472; ii. 17
    tertiary syphilis of, ii. 529
Nettle-fever, ii. 164
Nettle rash, ii. 164
Neuralgia, i. 513
  articular, i. 525
  cervicobrachial, i. 520
  cervico-occipital, i. 519
  crural, i. 521
  dorso-intercostal, i. 520
    anesthesia with, i. 521
    hyperesthesia with, i. 521
  glossalgia, i. 519
  in aortic aneurysm, i. 69
  infraorbital, i. 518
  lingual, i. 519
  lumbo-abdominal, i. 521
  malarial form, i. 514
  neuritic, i. 514
  obturator, i. 522
  occipital, i. 519
  of coccyx, i. 524
  of external cutaneous nerves of thigh, i. 522
  of glans penis, i. 524
  of great auricular nerve, i. 519
  of inferior subcutaneous nerve of neck, i. 519
  of labia majora, i. 524
  of mammary gland, i. 521
  of penis, i. 524
  of perineum, i. 524

Neuralgia of scrotum, i. 524
  of supraclavicular nerve, i. 520
  of urethra, i. 524
  phrenic, i. 520
  sciatic, i. 522. See also *Sciatica.*
  spermatic, i. 524
  supraorbital, i. 518
  syphilis and, i. 514
  treatment, i. 519
  trigeminal, i. 516
  varieties, i. 514
Neurasthenia, ii. 144
  diagnosis, ii. 148
  treatment, ii. 148
Neuritis, i. 531
  apoplectiform, i. 532
  ascending, i. 534
  hemorrhagic, i. 532
  interstitial, i. 532
  migratory, i. 532
  mixed, i. 532
  multiple, i. 534
    paralysis with, i. 535
    recurrent, i. 536
  nodose, i. 532
  parenchymatous, i. 532
  segmental, i. 533
  suppurative, i. 532
  toxic, i. 536
  treatment, i. 534
Neuron, i. 542
  cerebrospinal, i. 543; ii. 32
  of second degree, i. 543
Neuroses, cardiac, i. 61
  central, ii. 86
    with motor disturbances predominant, ii. 87
    with psychic alterations predominant, ii. 139
    with sensory disturbances predominant, ii. 115
    with trophic disturbances predominant, ii. 120
    with vasomotor disturbances predominant, ii. 120
  cutaneous, ii. 209
  occupation-, ii. 98
  traumatic, ii. 149
    shock and, ii. 149
    spinal symptoms, ii. 150
    treatment, ii. 152
  vasomotor articular, intermittent, ii. 121
    of extremities, ii. 120
Neutrophile cells, ii. 240
Nevus, ii. 199
Newborn, meléna of, i. 288
Nictitation, i. 508
Nigrities, ii. 219
  syphilitic, ii. 515
Nocturnal pollution, i. 466
Nodes, Heberden's, ii. 308
  singers', i. 82
Nodose erythema, ii. 166
Nodules, gouty, ii. 281

Nose, catarrh of, chronic atrophic, i. 75
    hypertrophic, i. 75
    complications, i. 74
    ozena with, i. 76
    treatment, i. 76
  diseases of, i. 73
  eczema of, ii. 172
  tertiary syphilis of, ii. 524
Nucha, eczema of, ii. 172
Nucleo-albumins in urine, i. 389
Nun's murmur, i. 313
Nutmeg-liver, i. 323, 324
  cyanotic, i. 23
Nylander's test for sugar in urine, ii. 288

OBERMEIER'S spirilla, ii. 358
Obesity, ii. 275
  drinking-cures for, ii. 279
  treatment, ii. 278
Obstetric paralysis, i. 499
Obturator nerve, peripheral paralysis, i. 505
Occupation-neuroses, ii. 98
Oertel's treatment of obesity, ii. 278
Œsophagomycosis oïdica, i. 203
Oïdium albicans, i. 180
  Schoenleinii, ii. 222
Ointment, variolous, ii. 347
Olfactory nerve, disease of, i. 529
Oligosteatosis, ii. 197
Onychauxis, ii. 203
Onychomycosis favosa, ii. 225
Ophthalmoplegia, chronic progressive, ii. 25
  external nuclear, ii. 25
Optic thalamus, diseases of, ii. 38
Orchitis in mumps, ii. 386
Osmidrosis, ii. 193, 195
Osteitis, deforming, ii. 138
Osteo-arthropathy, hypertrophic, ii. 138
Osteomalacia, ii. 305
Osteopsathyrosis, syphilitic, ii. 524
Oxaluria, ii. 299
Ox-heart, i. 27
Oxyuris vermicularis, i. 303
Ozena with chronic coryza, i. 76

PACHYMENINGITIS, cerebral, internal hemorrhagic, ii. 82
  spinal, external, i. 601
    internal, i. 602
Pain-sense, testing of, i. 526
Palate, soft, catarrh of, i. 184. See also *Angina.*
    diseases of, i. 184
    paralysis of muscles of, ii. 22
  tertiary syphilis of, ii. 526
Palsy, bandage-, i. 492
  coachmen's, i. 492
  crutch-, i. 492
  prisoners', i. 492
  water-carriers', i. 492
Pancreas, calculi in ducts of, i. 378
  carcinoma of, i. 392

- Pancreas, fat-necrosis in, i. 371
  - hemorrhage into, i. 371
  - inflammation of, i. 371
  - tertiary syphilis of, ii. 528
- Pancreatitis, i. 371
- Panum's test for albumin, i. 389
- Papillitis and brain tumors, ii. 70
- Parageusia, i. 531
- Paralysis, acute cerebral, of childhood, ii. 67
    - spinal, of childhood, i. 583
  - agitans, ii. 108
    - "pen-holder" position in, ii. 111
    - treatment, ii. 111
  - alcoholic, i. 539
  - arsenical, i. 531
  - bulbar. See *Bulbar paralysis.*
  - compression-, spinal, i. 567
    - general paralysis with, i. 569
    - treatment, i. 571
  - drummers', i. 493
  - fright-, i. 601
  - from aortic aneurysm, i. 69
  - from ergot-poisoning, i. 539
  - lead-, i. 536
  - motor, of trigeminal nerve, i, 476
  - narcosis-, i. 493
  - obstetric, i. 499
  - of axillary nerve, i. 498
  - of broad dorsal muscle, i. 503
  - of cerebral nerves, multiple, i. 490
    - syphilis and, ii. 531
  - of cervical sympathetic, ii. 85
  - of corrugator muscle of eyebrow, i. 481
  - of depressor of epiglottis, ii. 22
  - of diaphragm, i. 491
  - of elevator of angle of scapula, i. 503
  - of external rotators of arm, i. 504
  - of facial nerve, i. 477. See also *Facial nerve, paralysis of.*
  - of hypoglossal nerve, i. 489
  - of iliopsoas muscle, i. 505
  - of internal rotators of arm, i. 503
  - of median nerve, i. 495
  - of muscles of auricle, i. 482
    - of esophagus, ii. 23
    - of face, i. 478
    - of mastication, i. 476
    - of soft palate, ii, 22
    - of vocal band, ii. 23
  - of musculocutaneous nerve, i. 498
  - of nerves of arm and brachial plexus, i. 498
  - of orbicular muscle of eyelids, i. 481
  - of pectoral muscles, i. 503
  - of phrenic nerve, i. 490
  - of platysma myoides, i. 482
  - of radial nerve, i 492
  - of rhomboid, i. 503
  - of serrate muscle, i. 500
  - of spinal accessory nerve, i. 488
    - nerve-roots, i. 602
  - of sternocleidomastoid muscle, unilateral, i. 488
  - of tibial nerve, i. 506
  - of tongue, i. 489
  - of trapezius muscle, i. 488, 489
- Paralysis of ulnar nerve, i. 497
  - periodic, i. 507
  - peripheral, i. 472
    - muscular contractions in, i. 473
    - of abdominal muscles, i. 504
    - of crural nerve, i. 505
    - of gluteal nerves, i. 506
    - of muscles of back, i. 504
    - of obturator nerve, i. 505
    - of scapular muscles, i. 500
    - of sciatic nerve, i. 506
    - treatment, i. 475
  - pressure-, i. 476
  - pseudobulbar, ii. 24
  - psychic, i. 601
  - reflex, i. 600
  - saturnine, i. 536
  - sleep-, i. 492
  - spinal, i. 546
    - acute ascending, i. 599
    - spastic, i. 581
  - temporary, i. 584, 586
  - uremic, i. 397
- Paramyoclonus, multiple, ii. 107
- Paranephritis, i. 432
- Paraphasia, ii. 40, 44, 45
- Pararenal connective tissue, inflam tion of, i. 432
- Parasteatosis, ii. 198
- Paratyphlitis, i. 257, 259
  - diagnosis, i. 263
  - symptoms, i. 263
  - treatment, i. 266
- Paridrosis, ii. 195
- Parosmia, i. 530
- Parotitis, epidemic, ii. 384
    - orchitis in, ii. 386
- Pectoral muscles, paralysis of, i. 503
- Pectus carinatum, ii. 302
  - gallinaceum, ii. 302
- Pediatrophy, syphilitic, ii. 533
- Pediculi, ii. 216
- Pediculi-salve, ii. 218
- Pediculus capitis, ii. 216
  - pubis, ii. 218
  - vestimentorum, ii. 219
- Pellagra, ii. 169
  - paralysis from, i. 539
- Pemphigus, ii. 176
  - acute, of newborn, ii. 176
  - syphilitic, ii. 515
- Pen-holder position, ii. 111
- Penis, chorda, ii. 424
  - neuralgia of, i. 524
- Pepsin in gastric juice, i. 209
- Peptones in urine, i. 389
- Pericardial cavity, gas in, i. 58
  - friction, i. 54
  - synechiæ, i. 53
- Pericarditis, i. 52
  - and dilatation of heart, i. 57
  - circumscribed, i. 53
  - diagnosis, i. 55, 56
  - diffuse, i. 52
  - dry, i. 52
  - external, i. 53
  - hemorrhagic, i. 53

Pericarditis humida, i. 52, 53
  hyperirritability of myocardium after, i. 56
  myocardial weakness in, i. 55
  prognosis, i. 57
  purulent, i. 53
  putrid, i. 53
  serofibrinous, i. 53
  sicca, i. 52
  spontaneous, i. 52
  treatment, i. 57
  tuberculous, ii. 505
  with effusion, i. 52, 53
Pericardium, alterations of blood in, i. 60
  chyle in, i. 60
  diseases of, i. 52
  dropsy of, i. 59
  inflammation of, i. 52. See also *Pericarditis.*
  tumors of, i. 60
Perichondrium of larynx, inflammation of, i. 87
Pericystitis, i. 450
Perihepatitis, i. 325
Perimyelitis, i. 554
  chronic, i. 558
Perineum, neuralgia of, i. 524
Peripheral disease of nerves of special sense, i. 529
  nerves, degeneration of, i. 474
    diseases of, i. 472
    inflammatory and degenerative disorders of, i. 531. See also *Neuritis.*
    tertiary syphilis of, ii. 529
  paralysis, i. 472
    muscular contractions in, i. 473
    treatment, i. 475
Perisplenitis, ii. 233
Peritoneum, carcinoma of, i. 386
  diseases of, i, 372
  echinococcus of, i. 386
  inflammation of, i. 372. See also *Peritonitis.*
  tertiary syphilis of, ii. 523
Peritonitis, i. 372, 375-380
  alimentary, i. 373
  by extension, i. 373
  chronic, symptoms, i. 379
  cryptogenetic, i. 373
  diffuse, i. 374, 375
  fatty, i. 383
  fibrinous, i. 374
  hemorrhagic, i. 375
  obliterative, i. 374
  perforative, i. 373, 377
  purulent, i. 374, 375
  putrid, i. 375
  rheumatic, i. 372
  serous, i. 374, 379
  suppurative, i. 374
  symptoms, 375-380
  traumatic, i. 372
  treatment, i. 380
  tuberculous, ii. 502
    abdomen in, ii. 503, 504
Perityphlitis, i. 257
  complications, i. 261
  diagnosis, i. 263
  symptoms, i. 260
  treatment, i. 264
Pernicious anemia, progressive, ii. 250. See also *Anemia.*
Peroneal nerve, palsy of, i. 506
Pertussis, ii. 374
  diagnosis, ii. 378
  in pregnancy, ii. 378
  larynx in, ii. 376
  stomach in, ii. 377
  trachea in, ii. 376
  treatment, ii. 379
Pertussis-pneumonia, ii. 378
Pettenkofer's test for biliary coloring-matter, i. 319
Pharyngitis, acute phlegmonous, ii. 387
  catarrhal. i. 184
  chronic atrophic, i. 189
  circumscribed, i. 187
  complications, i. 187
  diffuse, i. 187
  granular, i. 188
  parenchymatous, i. 189
  superficial, i. 186, 188
  treatment, i. 189
Pharynx, catarrh of, i. 184
  chronic tuberculosis of, ii. 488
  diseases of, i. 184
  tertiary syphilis of, ii. 526
Phimosis, treatment, ii. 197
Phloroglucin-vanillin solution, Günzburg's, i. 207
Phosphaturia, ii. 299
Phrenic nerve, paralysis of, i. 490
Phthisical habitus, 471, 478
  treatment, ii. 485
Phthisis, florid pulmonary, ii. 480
  pituitosa, i. 101
  pulmonary, ii. 470. See also *Tuberculosis.*
  ventriculi, i. 213
Pica, i. 240
Piedra, ii. 208
Piitis, i. 603
Pilimictio, i. 458
Pituitary body, hyperplasia of, ii. 69
  struma, ii. 69
Pityriasis, simple, ii. 197
  rubra, ii. 189
  versicolor, ii. 221
Plague, ii. 371
  bacillus of, ii. 372
  treatment, ii. 374
Plague-pneumonia, ii. 373
Plasmodia of malaria, ii. 362
Platyhelminthes in intestine, i. 293
Platysma myoides, paralysis of, i. 482
Plethora and fat heart, i. 30
Pleura, cachectic edema of, i. 169
  carcinoma of, i. 170
  diseases of, i. 152
  dropsy of, i. 169
  echinococcus of, i. 171

Pleura, inflammation of, i. 152. See also *Pleurisy*.
  tertiary syphilis of, ii. 526
Pleural cavity, blood in, i. 170
    chyle in, i. 170
    hypostatic edema of, i. 168
  exudates, influence on other organs, i. 156
Pleurisy, i. 152
  auscultation in, i. 155
  cryptogenetic, i. 153
  diagnosis, i. 158
  diaphragmatic, i. 158
  dry, i. 153
  fibrinous, i. 153
  hemorrhagic, i. 153
  inspection in, i. 154
  interlobular, i. 158
  moist, i. 153
  multilocular, i. 158
  palpation in, i. 155
  percussion in, i. 155
  pulsating, i. 158
  purulent, i. 153
    rupture of pus in, i. 157
  serous, i. 153
  treatment, i. 160
  tuberculous, ii. 505
Pleuropneumonia, fibrinous, i. 130
Plica polonica, ii. 171, 217
Pneumatotherapy for pleurisy, i. 161
Pneumococcus of bronchopneumonia, i. 125
  of Fränkel, morphology, i. 128
Pneumonia, abortive, i. 134
  afebrile, i. 134
  aspiration-, i. 126
    in perichondritis of larynx, i. 88
  asthenic, i. 135
  bilious, i. 135
  catarrhal, i. 125
    treatment, i. 128
  croupous. i. 128. See also *Pneumonia, fibrinous*.
  dissecting, i. 140
  ephemeral, i. 134
  erratic, i. 135
  fibrinosa cruciati, i. 131
    duplex, i. 131
  fibrinous, i. 128
    anomalies, complications, and sequelæ. i. 134
    delirium in, i. 136
    diagnosis, i. 137
    incubation-period, i. 132
    of children, i. 135
    prune-juice sputum in, i. 136
    symptoms, i. 132
    treatment, i. 138
  foreign-body, i. 126
  gelatinous, ii. 474
  intermittent, i. 135
  interstitial, i. 139
    treatment, i. 142
  lobar, i. 130
  malignant, i. 135
  massive, i. 130
Pneumonia, measles-, ii. 320
  pertussis-, ii. 378
  plague-, ii. 373
  progressive, i. 134
  protracted, i. 134
  pulmonary abscess after, i. 142
  recurrent, i. 135
  serous, i. 124
  totalis, i. 131
  typhoid, i. 135
  wandering, i. 135
  white, ii. 535
  with influenza, ii. 382
Pneumopericardium, i. 58
Pneumoperitonitis, i. 375, 377
  sacculated, i. 378
Pneumothorax, i. 162
  closed, i. 167
  diagnosis, i. 166
  open, i. 167
  transitional, i. 167
  treatment, i. 168
  valvular, i. 167
Pneumotyphoid, ii. 394
Poikilocytosis, i. 313
Polio-encephalitis, acute infantile, ii. 67
  progressive inferior, ii. 20
    facial expression in, ii. 22
    treatment, ii. 24
  superior, ii. 24
Poliomyelitis, i. 554
  anterior, i. 474, 542, 582
  chronic, i. 558
  of adults, i. 586
Pollution, nocturnal, i. 466
    alarms for, i. 467
Polyarthritis, acute primary, ii. 349
    diagnosis, ii. 352
    treatment, ii. 352
  deforming, ii. 138
  scarlatinal, ii. 328
Polychromophilia, ii. 252
Polymyositis, ii. 161
Polyneuritis, i. 534
  paralysis with, i. 535
  recurrent, i. 536
Polysarcia, ii. 275
  treatment, ii. 278
Polyuria, ii. 296
Pons, disease of, focal symptoms in, ii. 37
  Varolii, disease of, ii. 37
Pontine symptoms, ii. 38
Porcupine-people, ii. 200
Porencephaly, ii. 68
Portal vein, purulent inflammation of, i. 370
    thrombosis of, i. 368
Post-epileptic state, ii. 90
Precordium, pains in, i. 63
Pre-epileptic insanity, ii. 89
Pregnancy, pertussis in, ii. 378
  typhoid fever in, ii. 390
Preparoxysmal insanity, ii. 89
Pressure-sense, testing of, i. 526
Presystolic murmur, i. 40
Proctitis, i. 244, 246
  chronic catarrhal, i. 255

Profeta's law, ii. 533
Propeptones in urine, i. 389
Prostatitis, gonorrheal, ii. 426
Prostatorrhea, i. 468
Protozoa in intestine, i. 292
Prune-juice sputum in pneumonia, i. 136
Prurigo, ii. 190
  bullous, ii. 190
  urticaria with, ii. 190
Pruritus, cutaneous, ii. 209
Pseudo-apoplectic attacks in fat heart, i. 30
Pseudobulbar paralysis, ii. 24
Pseudocroup, i. 81
Pseudoleukemia, ii. 247
  treatment, ii. 250
Pseudotabes, post-diphtheric, ii. 461
Psoriasis, ii. 186
  syphilitic, ii. 313
[illegible]
Ptyalism, i. 182
  false, i. 183
  idiopathic, i. 184
  primary, i. 184
Puerperal fever and endocarditis, i. 47
Pulex irritans, ii. 220
  penetrans, ii. 220
[illegible]
Pulmonary abscess, i. 162
  [illegible]
  [illegible]
  [illegible]
  [illegible] valvular disease, i. 42
  [illegible] valvular disease, i. 42
  [illegible]
  [illegible] valvular disease, i. 42
  [illegible] See also [illegible]
[illegible]
[illegible]

Pyramidal tract, direct, i. 545
  lateral, i. 543
Pyrosis, i. 211

QUADRIGEMINATE bodies, lesions in, ii. 38

RABIES, ii. 549
Rachitic rosary, ii. 302
Rachitis, ii. 299
  and spasm of glottis, i. 94
  chest in, ii. 301
  clavicles in, ii. 302
  diagnosis, ii. 303
  epiphyses in, ii. 302, 303
  of bones, ii. 300, 304
  of pelvic bones, ii. 302
  pathogenesis, ii. 304
  scapulæ in, ii. 302
  sternum in, ii. 302
  teeth in, ii. 301
  treatment, ii. 305
Radial nerve, paralysis of, i. [illegible]
Rag-sorters' disease, ii. 542
Rats, trichinosis, i. 306
Ray-fungus, ii. 546
Raynaud's disease, ii. 122
Reactionary fever, i. 507
Recurrent laryngeal nerves, paralysis of, i. 91, 92
  from carcinoma of œsophagus, i. 282
Regurgitation, nervous, i. 225
Relapsing fever, i. 257
  abscess of spleen in, i. 260
  [illegible]
  [illegible]
  [illegible]
  [illegible]
  treatment, i. 261
[illegible]
[illegible]
[illegible]

Rhomboid muscle, spasm of, i. 511
  paralysis of, i. 503
Robertson's symptom, i. 575
Roseola, syphilitic, ii. 514
Rötheln, ii. 322
Rubeola, ii. 322
Rumination, i. 237
Rupia, syphilitic, ii. 523

SAGO-SPLEEN, ii. 234
Saline discharge, ii. 173
Salivation, i. 182
Saltatory spasm, ii. 107
Sand, cutaneous, ii. 198
Sarcocele, syphilitic, ii. 528
Sarcoptes hominis, ii. 211
Satiety, disorders of sense of, i. 240
Saturnine paralysis, i. 536
Scabies, ii. 211
  Norwegian, ii. 215
  treatment, ii. 215
Scalp, eczema of, ii. 171
  herpes tonsurans of, ii. 227
  seborrhea of, ii. 195
Scanning speech, i. 561
Scapula, elevator of angle of, paralysis of, i. 503
  spasm of, i. 511
Scapular muscles, peripheral paralysis of, i. 500
Scarlet fever, ii. 323
  and verrucose endocarditis, i. 50
  anomalies, ii. 326
  complications, ii. 327
  diagnosis, ii. 329
  eruption of, ii. 325
  febrile albuminuria in, ii. 328
  temperature-curve, ii. 325
  tongue in, ii. 326
  treatment, ii. 329
Schistocytes, ii. 252
Sciatic nerve, peripheral paralysis of, i. 506
Sciatica, i. 522
  ascending, i. 523
  descending, i. 523
  neuritic, i. 523
  scoliotic, i. 523
    sugar in urine in, i. 523
Sclerema of newborn, ii. 203
Sclerodactyly, ii. 126
Scleroderma, ii. 126
Sclerosis, amyotrophic lateral, i. 593
    treatment, i. 596
  cerebrospinal, multiple, i. 560
    nystagmus in, i. 562
    treatment, i. 563
Scorbutic gingivitis, ii. 266, 267
Scorbutus, ii. 266
  hemorrhages in, ii. 267
Scotomata, fortification-, ii. 119
Scriveners' palsy, ii. 98
Scrofulosis, ii. 506
  erethetic, ii. 507
  facies in, ii. 507
  torpid, ii. 507
  treatment, ii. 509
Scrofulous habitus, ii. 507
  lichen of, ii. 191
Scrotum, neuralgia of, i. 524
Scurvy, ii. 266
  hemorrhages in, ii. 267
Sea-scurvy, ii. 266
Sebaceous glands, secretory disorders of, ii. 195
  secretion, diminished, ii. 197
    disorders of, ii. 198
    increased, ii. 195
Seborrhea, ii. 195
  universal, ii. 197
Seminal fluid, involuntary discharge of, i. 465. See also *Spermatorrhea.*
Semi-ovale, centrum, diseases of, focal symptoms in, ii. 34
Sensibility, compasses for testing, i. 526
  cutaneous, i. 525
Sensory nerves, inflammation of, i. 533
Seropneumopericardium, i. 58
Seropneumothorax, i. 162
Serrate muscle, paralysis of, i. 500
Serum, antidiphtheric, ii. 462, 469
  tetanus, ii. 455
Sexual organs, male, diseases of, i. 463
Shaking palsy, ii. 108
Shock and traumatic neuroses, ii. 149
Sialodochitis, fibrinous, i. 184
Singers' nodes, i. 82
Singultus, i. 211
Siphon, stomach-, soft rubber, i. 232
Situs viscerum perversus s. inversus, i. 34
Skin, angioneurosis of, ii. 166
  atrophy of, ii. 204
  discolorations of, ii. 200
  diseases of, ii. 164
  electric sensibility of, i. 526
  erysipelas of, ii. 332
  hemorrhage into, ii. 262
  herpes tonsurans of, ii. 227
    zoster of, ii. 335
  hypertrophies of, ii. 199
  inflammations of, ii. 164
    bullous, ii. 176
    erythematous, ii. 164
    papular, ii. 190
    pustular, ii. 179
    scaly, ii. 186
    vesicular, ii. 170
  itching of, ii. 209
  parasites of, ii. 211
    animal, ii. 211
    vegetable, ii. 221
  pigment of, atrophy of, ii. 204
    hypertrophies of, ii. 199
  secretory disorders of, ii. 193
  tertiary syphilis of, ii. 522
Skull, floor of, ii. 41
Slaty induration, i. 140
Sleep-paralysis, i. 492
Small-pox, ii. 341
  abortive, ii. 346
  confluent, ii. 346
  conjunctiva in, ii. 345
  diagnosis, ii. 346

Small-pox, eruption of, ii. 343
  esophagus in, ii. 345
  hemorrhagic, ii. 346
  immunity from, ii. 342, 348
  pustule of, ii. 347
  stomach in, ii. 345
  temperature in, ii. 345
  temperature-curve, ii. 343
  treatment, ii. 348
Spanish collar, ii. 424
Spasm, muscular, i. 507
  rotatory, i. 511
Spasmodic tic, ii. 108
Spasmus glottidis ablactatorum, i. 94
Spastic rigidity of extremities, i. 581
Spastic-paretic gait, i. 581, 582
Special sense, nerves of, peripheral disease of, i. 529
Speech, acquisition of, diagrams, ii. 43
  disorders of, ii. 43
  scanning, i. 561
Spermatocystitis, gonorrheal, ii. 426
Spermatorrhea, i. 465
  charlatans' treatment, i. 467
  permanent, i. 466
  true, i. 466
Spinal accessory nerve, paralysis of, i. 488
    spasm, i. 510
  bulb, ii. 17
  column and dura mater, relation of, i. 547
  compression-paralysis, i. 567
    general paralysis with, i. 569
    treatment, i. 571
  cord, abscess of, i. 553
    acute inflammation of, i. 552. See also *Myelitis, acute.*
    anatomy, i. 540
    anemia of, i. 547
    anterior horns of, i. 542
      circumscribed hemorrhage into, i. 551
    cavities in, i. 564
    chronic inflammation of, i. 558
    columns of, diagram of, i. 544
    diseases of, i. 540
      asystematic, i. 547
      atypical, i. 547
      combined system, i. 592
      divisions of, i. 547
      rules for diagnosis, i. 541
      symptoms, i. 546
      system, i. 572
      typical, i. 572
    functional disorders of, i. 599
    gray matter of, i. 541
    hemorrhages into, i. 548
      circumscribed, i. 550
      treatment, i. 551
      tubular, i. 550
    hyperemia of, i. 548
    in hereditary ataxia, i. 595
    neuroses of, i. 599
    physiology, i. 540
    secondary degeneration of, i. 596–599
    single system-diseases of, i. 572
Spinal cord, soft membranes of, inflammation of, i. 603–606
      softening of, i. 553
      syphilis of, ii. 529
      systems of, i. 542
      transverse lesion of, diagnosis, i. 570
        section of, i. 542
      tumors of, i. 564
      unilateral lesions of, i. 571
      white matter of, i. 541
          columns of, i. 543
    dura mater, inflammation of, i. 601
    irritation, ii. 146
    membranes, hemorrhage into, i. 607
    meninges, diseases of, i. 601
      neoplasms of, i. 609
    meningitis, i. 603
      acute, i. 603
        treatment, i. 606
      chronic, i. 606
    pachymeningitis, external, i. 601
      internal, i. 602
    paralysis, i. 546
      acute ascending, i. 599
        of childhood, i. 583
      spastic, i. 581
    progressive muscular atrophy, i. 588
        treatment, i. 592
    sclerosis, posterior, i. 572. See also *Tabes dorsalis.*
    softening, i. 553
Spirilla of relapsing fever, ii. 358
Spleen, abscess of, ii. 231
  amyloid, ii. 234
  capsule of, inflammation of, ii. 233
  carcinoma of, ii. 235
  diseases of, ii. 230
  displacement of, ii. 237
  echinococcus of, ii. 235
  embolic infarction of, 230
  ham-, ii. 234
  in leukemia, ii. 242
  infection, acute, i. 131
  movable, ii. 236
  rupture of, ii. 236
  sago-, ii. 234
  sarcoma of, ii. 235
  tertiary syphilis of, ii. 528
  wandering, ii. 236
Splenic artery, aneurysm of, ii. 238
  embolism of, i. 43
Splenitis, ii. 233
Splenius muscle of head, spasm of, i. 511
Sporadic cretinism, ii. 128
Sputa globosa fundum appetentia, ii. 478
Sputum, elastic fibers in, in tuberculosis, ii. 476
  globose, ii. 478
  globular, i. 100
  in tuberculosis, ii. 477
  nummular, ii. 478
  prune-juice, in pneumonia, i. 136
  tubercle bacilli in, ii. 475
Sputum-cocci, i. 129
Sputum-septicemia, cocci of, i. 129
Stasis, urine of, i. 19
Status epilepticus, ii. 90

Stellwag's symptom, ii. 133
Stenocardia, i. 63
Stenosis, aortic, with valvular disease, i. 38
Sterility after gonorrheal urethritis in male, ii. 425
  in male, i. 464
Sternocleidomastoid muscle, unilateral paralysis of, i. 488
Sternomastoid, spasm of, i. 510
Stimulants in cardiac insufficiency, i. 24
Stomach, absorptive power of, i. 205
  ante-, i. 199
  atony of, i. 238
  carcinoma of, i. 222
    treatment, i. 226
  dilatation of, i. 227
    treatment, i. 231
    yeast-cells and sarcinæ from vomitus of, i. 230
  diseases of, i. 205
  displacements of, i. 233
  entire, tonic spasm of, i. 236
  fungus of, i. 223
  hypermotility of, i. 236
  in small-pox, ii. 345
  motor activity of, i. 206
  musculature of, tonic spasm of, i. 236
  nervous tormina of, i. 236
  neuroses of, i. 234
    mixed, i. 241
    motor, i. 234
    secretory, i. 240
    sensory, i. 238
  peristaltic unrest of, i. 236
  suppurative inflammation of, i. 216
  tertiary syphilis of, ii. 527
  ulcer of, latent, i. 218
    round, i. 217
      treatment, i. 220
Stomach-contents, acetic acid in, i. 209
  acidity of, i. 207, 208
  butyric acid in, i. 209
  examination of, i. 207
  hydrochloric acid in, i. 207
  lactic acid in, i. 209
  organic acids in, i. 209
Stomach-siphon, soft-rubber, i. 232
Stomatitis, aphthous, i. 178
  catarrhal, i. 175
  oïdica, i. 179
  ulcerative, i. 177
Stomatomycosis sarcinica, i. 181
Streptococcus erysipelatosus in skin, ii. 331
Striate body, ii. 36
Stringhalt gait, i. 577
Stroke, ii. 55
Strawberry-tongue, ii. 326
Struma, substernal, i. 172
St. Vitus's dance, ii. 100
    diagnosis, ii. 104
    treatment, ii. 104
Subacidity, i. 240
Subarachnoid hemorrhage, i. 608; ii. 84
Subdural hemorrhage, i. 608; ii. 84
Subpial hemorrhage, i. 608
Substantia nigra, ii. 36
Subsultus tendinum in typhoid, ii. 394
Suffocation in perichondritis of larynx, i. 88
Sugar in urine, quantitative determination, ii. 289
    tests for, ii. 287
Superacidity, i. 240
Superior longitudinal sinus, thrombosis of, ii. 81
Supraclavicular nerve, neuralgia of, i. 520
Swallowing, disorders of, ii. 19
Swamp-fever, ii. 361. See also *Malarial fever.*
Sweat, alterations in, ii. 195
  diminished secretion of, ii. 194
  increased secretion of, ii. 193
Sweat-glands, secretory disorders of, ii. 193
Sweating, axillary, ii. 193
  blood-, ii. 142, 195
  of feet, ii. 194
  of hands, ii. 194
  sickness, English, ii. 338
  unilateral, ii. 193
Sycosis, ii. 182
Sydenham's chorea, ii. 100
    diagnosis, ii. 104
    treatment, ii. 104
Sylvian fossa, artery of, embolism of, ii. 62
Sympathetic, cervical, irritation of, ii. 86
    paralysis of, ii. 85
  nerve, diseases of, ii. 85
Syncope, total, in pleurisy, i. 157
Syphilids, ii. 511, 514
  diagnosis, ii. 517
  in hereditary syphilis, ii. 534
Syphilis, ii. 510
  acquired, ii. 510
    diagnosis, ii. 517
    treatment, ii. 518
  alopecia in, ii. 515
  broad condylomata in, ii. 514, 516
  buboes in, ii. 513
  diagnosis, ii. 517
  hemorrhagic, ii. 518
  hereditary, ii. 532
    anatomic changes, ii. 535
    Hutchinson's teeth in, ii. 534
    late, ii. 533
    syphilids in, ii. 534
    tertiary symptoms in, ii. 534
    treatment, ii. 536
  infection with, ii. 510, 511
  lichen of, ii. 515
  malignant, ii. 518
  mercurialism and, ii. 520
  mucous membranes in, ii. 516
  primary, ii. 510
    treatment, ii. 518
  second period of incubation, ii. 513
  secondary, ii. 510, 514
    treatment, ii. 521
  syphilids of, ii. 514

Syphilis, tertiary, ii. 516, 522-532
    treatment, ii. 521, 522
  treatment, ii. 518
    inunction, ii. 519
  vaccination and, ii. 511
  visceral stage, ii. 516
Syringomyelia, i. 564
  anesthesia with, i. 566
  atrophica, i. 566

TABES dorsalis, i. 572
    absence of knee-jerk in, i. 575
    ataxia in, i. 576
    cervical, i. 575
    course of, i. 579
    cutaneous anesthesia in, i. 577
    diagnosis, i. 580
    gastric crises in, i. 578
    gymnastics for, i. 581
    intestinal crises in, i. 579
    paralysis with, i. 579
    spinal nerve-roots in, i. 574
    syphilis and, i. 572; ii. 529
    treatment, i. 580
  mesenteric, ii. 508
Tachycardia, paroxysmal, i. 61
Tactile sensibility, i. 525
Tænia echinococcus, i. 350
  mediocanellata s. saginata, i. 293
  saginata, i. 297
  solium, i. 293, 299
Tapeworms, i. 293
  heads of, i. 299
  ova of, i. 295
  proglottides of, i. 295
  treatment, i. 298
Tegmentum, ii. 36
  disease of, ii. 37
Temperature-sense, testing of, i. 526
Tendon-sheaths, gout of, ii. 281
Tenesmus, anal, i. 246; ii. 405
Test, Boedecker's, for albuminuria, i. 389
  boiling-nitric-acid, i. 388
  fermentation-, for sugar, ii. 288
  for acidity of gastric contents, i. 208
  for albuminuria, i. 388
  for sugar in urine, ii. 287
  four-glass, i. 210
  Galippe's picric-acid, i. 389
  Gmelin's, for biliary coloring-matter, i. 319
  Heller's, for hematuria, i. 393
    nitric-acid, i. 389
  Jaffe's, for indican in urine, i. 281
  Maréchal's, for biliary coloring-matter, i. 319
  Moore's, for sugar, ii. 287
  Nylander's, for sugar, ii. 288
  Panum's, for albumin, i. 389
  Pettenkofer's, for biliary coloring-matter, i. 319
  Trommer's, for sugar, ii. 288
  two-glass, ii. 429
Test-breakfast, i. 207
Testicle, inflammation of, in mumps, ii. 386
Testicle, tertiary syphilis of, ii. 528
Test-meal, i. 206
Tetanus, ii. 449
  antitoxin for, ii. 455
  bacillus of, ii. 449
  diagnosis, ii. 454
  facies in, ii. 453
  localized, ii. 454
  serum for, ii. 455
  treatment, ii. 454
Tetany, ii. 94
  and laryngismus stridulus, ii. 97
  Chvostek's facial phenomenon in,
  thyroid gland and, ii. 95
  transverse phenomenon in, ii. 97
Tetter, moist, ii. 170
Thalamus, optic, disease of, ii. 38
Thigh, external cutaneous nerve neuralgia of, i. 522
  pseudohypertrophy of extensor ii. 155
Thomsen's disease, ii. 162
Thorax, barrel-like, in emphysem lungs, i. 118
  examination of, in bronchial ast i. 114
  paralytic, ii. 479
  permanent expiratory, ii. 479
Thread-test, Garrod's, ii. 282
Thrombophlebitis, treatment, ii. 82
Thrush-fungus, i. 180
Thyro-ary-epiglottic muscles, par of, i. 92
Thyro-arytenoid muscles, paralysi i. 90
Tibial nerve, paralysis of, i. 506
Tic, rotatory, i. 511
Tickling-sense, testing of, i. 526
Time-sense, testing of, i. 526
Tongue, black, i. 182
  geographical, i. 182
  hairy, i. 182
  paralysis of, i. 489
  spasm of, i. 510
  strawberry, ii. 326
  tertiary syphilis of, ii. 526
Tophi, arthritic, ii. 281
Tormina, nervous intestinal, i. 290
    of stomach, i. 236
Torticollis, i. 605
Trabecular degeneration, i. 107
Trachea, diseases of, i. 96
  tertiary syphilis of, ii. 525
Transverse sinus, thrombosis of, ii.
Trapezius muscle, paralysis of, i. 489
  spasm of, i. 510
Tremor, ii. 112
  intention-, i. 561
Trichina spiralis, i. 305
Trichinæ, muscle-, i. 305
Trichiniasis, i. 305
  diagnosis, i. 310
  symptoms, i. 308
  treatment, i. 310
Trichocephalus dispar, i. 304
Trichomonas intestinalis, i. 293

Trichophyton onychomycosis, ii. 228
Trichophyton-fungi, ii. 227
Trichorrhexis, ii. 207
Tricuspid insufficiency with valvular disease, i. 40
obstruction with valvular disease, i. 41
stenosis with valvular disease, i. 41
Trigeminal, motor spasm of, i. 507
nerve, distribution of, i. 518
motor paralysis of, i. 476
Trismus, ii. 452
Trommer's test for sugar in urine, ii. 288
Trousseau phenomenon in tetany, ii. 97
Tubercle bacillus in sputum, ii. 475
in urine, ii. 493
infection with, ii. 471
Tuberculosis, ii. 470
bacillus of. See *Tubercle bacillus.*
chronic intestinal, ii. 488
laryngeal, ii. 486
of bladder, ii. 491
treatment, ii. 495
of kidneys, ii. 491
treatment, ii. 495
pharyngeal, ii. 488
pulmonary, ii. 470
auscultatory phenomena in, ii. 480
complications, ii. 481
diagnosis, ii. 475
elastic fibers in, ii. 476
examination of lungs in, ii. 479
night-sweats in, ii. 480
pneumothorax in, ii. 481
sputum in, ii. 477
stomach in, ii. 480
symptoms, ii. 475
treatment, ii. 483
general miliary, ii. 495
treatment, ii. 498
of pelvis of kidney, ii. 492
solitary, ii. 510
urogenital, ii. 491
Tuberculous caverns, ii. 474
ulcers of intestine, ii. 490
Tunnel-anemia, i. 311
Two-glass test, ii. 429
Typhlitis, i. 257
diagnosis, i. 263
stercoral, i. 259
symptoms, i. 262
treatment, i. 265
Typhoid, bilious, ii. 360
cholera-, ii. 416
fever, ii. 387
abdomen in, ii. 392
abortive, ii. 393
afebrile, ii. 394
ambulatory, ii. 394
bacilli of, ii. 388
bowels in, ii. 393
Typhoid fever in pregnancy, ii. 390
intestinal hemorrhage in, ii. 396
lymph-follicles in, ii. 398
media of infection, ii. 388, 389
mesenteric glands in, ii. 399
myocardium in, ii. 395
perforative peritonitis in, ii. 396
post-mortem conditions, ii. 400
respiratory organs in, ii. 397
roseolæ of, ii. 392
sequelæ, ii. 397
spleen in, ii. 393, 400
stool in, ii. 393
subsultus tendinum in, ii. 394
symptoms, ii. 390
temperature-curve, ii. 391
thrombosis in, ii. 395
treatment, ii. 400
Widal reaction for, ii. 388
triangle, ii. 392
ulcer, ii. 399
Typhus, famine-, ii. 312
fever, ii. 311
crisis of, ii. 313
diagnosis, ii. 314
paralysis of heart in, ii. 313
rash of, ii. 313
temperature-curve, ii. 312
treatment, ii. 314
war-, ii. 312

UFFELMANN'S reagent, i. 209
Ulcer, fecal, i. 278
gouty, ii. 282
perforating, ii. 123
stercoral, i. 278
Ulnar nerve, paralysis of, i. 497
Umbilicus, eczema of, ii. 172
Uncinaria duodenalis, i. 311
Upper extremities, muscular spasm in, i. 513
Uremia, i. 396
treatment, i. 400
Uremides, i. 398
Ureter, carcinoma of, i. 447
diseases of, i. 435
tuberculosis of, ii. 494
Urethra, neuralgia of, i. 524
Urethritis, acute gonorrheal, in male, ii. 423
diagnosis, ii. 427
sterility after, ii. 425
treatment, ii. 433, 435
chronic gonorrheal, in male, ii. 428
gonorrheal, ii. 422
granular, ii. 432
ulcerative, ii. 432
Uridrosis, i. 398; ii. 195, 416
Urinary bladder. See *Bladder, urinary.*
organs, tuberculosis of, ii. 491
treatment, ii. 495

Urine in diabetes insipidus, ii. 297
    mellitus, ii. 287-290
  indican in, Jaffe's test for, i. 281
  nucleo-albumins in, i. 389
  of stasis, i. 19
  peptones in, i. 389
  propeptones in, i. 389
  sugar in, in scoliotic sciatica, i. 523
    quantitative determination, ii. 289
    tests for, ii. 287
  tubercle bacilli in, ii. 493
  unconscious evacuation of, i. 458
Urocystitis, i. 448
  acute, i. 450
  chronic, i. 452
  croupous, i, 450
  gonorrheal, ii. 426
  hemorrhagic, i. 451
  mucous, i. 451
  pseudodiphtheric, i. 450
  pseudomembranous, i. 450
  suppurative, i. 451
  treatment, i. 453
Urticaria, ii. 164

VACCINATION, ii. 342, 348
  syphilis and, ii. 511
Vagus, neurosis of, i. 113
Valvular disease, acquired, i. 35
  insufficiency, relative, i. 35
Varicella, ii. 338
  syphilitic, ii. 515
Variola, ii. 341. See also *Small-pox.*
Varioloid, ii. 345
Vasomotor articular neuroses, intermittent, ii. 121
  neuroses of extremities, ii. 120
Veins, gummata of, ii. 529
Venereal crown, ii. 515
Ventricles, dilatation and hypertrophy of, i. 24
      diagnosis and symptoms, i. 26
  hypertrophy of, i. 29
Vermiform appendix, inflammation of, i. 257. See also *Appendicitis.*
Vertebra prominens, i. 540
Vertebral columns, syphilis of, ii. 530
Vertigo, ii. 112
  electric, ii. 113
  epileptic, ii. 91
  essential, ii. 113
  gastric, i. 214
Vesicular column, i. 543
Viscera, internal, gout of, ii. 282
Vitiligo, ii. 204
Vitiligo, syphilitic, ii. 515
Vocal bands, muscles of, paralysis of, ii. 23
Volvulus, i. 277
Vomiting, blood, in newborn, i. 288
  fecal, i. 273, 280
  morning, of drunkards, i. 214
  nervous, i. 234
  periodic, i. 242

WANDERING kidney, i. 264
  spleen, ii. 236
War-scurvy, ii. 266
War-typhus, ii. 312
Waxy flexibility of muscles, ii. 114
Weaning, spasm of glottis and, i. 94
Weil's disease, ii. 420
Welker's blood-sedimenting method, ii. 240
Westphal's symptom in tabes dorsalis, i. 575
Whip-worm, ii. 221
White matter of spinal cord, i. 541
Whooping-cough, ii. 374. See also *Pertussis.*
Widal reaction, ii. 388
Williams's tracheal note, i. 155
Willisian hyperacusis, i. 482
Wintrich's change in pitch, i. 164
Word-blindness, ii. 34
Word-deafness, ii. 33, 39, 40
Worms, flat, sucking, i. 301
    tapeworms, i. 293
  of intestine, i. 293.
  round, i. 301
    Anguillula intestinalis, i. 315
      stercoralis, i. 315
    Ankylostomum duodenale, i. 311
    seat-, i. 303
    spool-, i. 301
    Trichina spiralis, i. 305
    whip-, i. 304
Writers' cramp, ii. 98
Wry-neck, i. 605

XANTHOPSIA, i. 320
Xeroderma, pigmented, ii. 209
  simple, ii. 209

YEAST-CELLS and sarcinæ from vomitus of gastric dilatation, i. 230
Yellow vision, i. 320

ZOONOSES, ii. 541

atalogue of the Medical Publications

OF

V. B. SAUNDERS & COMPANY

ILADELPHIA LONDON, W. C.
5 Walnut Street 161 Strand

rranged Alphabetically and Classified under Subjects

e page 18 for a List of Contents classified according to subjects

HE books advertised in this Catalogue as being *sold by subscription* are usually to be obtained from travelling solicitors, but they will be sent direct from the office of publication (charges of shipment prepaid) upon receipt of the prices given. All the other books vertised are commonly for sale by booksellers in all parts of the United States; but books l be sent to any address, carriage prepaid, on receipt of the published price.

Money may be sent at the risk of the publisher in either of the following ways: A postal ney order, an express money order, a bank check, and in a registered letter. Money sent any other way is at the risk of the sender.

ECIAL FFER To physicians of approved credit books will be sent, post-paid, on the following terms: $5.00 cash upon delivery of books, and monthly payments of $5.00 there-er until full amount is paid. Physicians not known to the house will be expected to furnish names of satisfactory references. Any one or two volumes will be sent on thirty days' e to those who do not care to make a larger purchase.

n American Text-Book of Applied Therapeutics.

Edited by James C. Wilson, M. D., Professor of Practice of Medicine and of Clinical Medicine, Jefferson Medical College, Philadelphia. Handsome imperial octavo volume of 1326 pages. Illustrated. Cloth, $7.00 net; Sheep or Half Morocco, $8.00 net. *Sold by Subscription.*

n American Text-Book of the Diseases of Children.

Second Edition, Revised.

Edited by Louis Starr, M. D., Consulting Pediatrist to the Maternity Hospital, etc.: assisted by Thompson S. Westcott, M. D., Attending Physician to the Dispensary for Diseases of Children, Hospital of the University of Pennsylvania. Handsome imperial octavo volume of 1244 pages, profusely illustrated. Cloth, $7.00 net; Sheep or Half Morocco, $8.00 net. *Sold by Subscription.*

n American Text-Book of Diseases of the Eye, Ear, Nose, and Throat.

Edited by G. E. de Schweinitz, M. D., Professor of Ophthalmology, Jefferson Medical College, Philadelphia; and B. Alexander Randall, M. D., Professor of Diseases of the Ear, University of Pennsylvania. Imperial octavo, 1251 pages, [illegible] of them in colors. Cloth, $7.00 net; Sheep or Half Morocco, [illegible] net. *Sold by Subscription*

An American Text-Book *of* Genito-Urinary *and* Ski Diseases.

Edited by L. Bolton Bangs, M. D., Professor of Genito-Urinary Su gery, University and Bellevue Hospital Medical College, New Yor and W. A. Hardaway, M. D., Professor of Diseases of the Skin, Mi souri Medical College. Imperial octavo volume of 1229 pages, with 3 engravings and 20 full-page colored plates. Cloth, $7.00 net; Sheep Half Morocco, $8.00 net. *Sold by Subscription.*

An American Text-Book *of* Gynecology, Medical *an* Surgical. Second Edition, Revised.

Edited by J. M. Baldy, M. D., Professor of Gynecology, Philadelphia Polyclinic, etc. Handsome imperial octavo volume of 718 pages; 341 illustrations in the text, and 38 colored and half-tone plates. Cloth, $6.00 net; Sheep or Half Morocco, $7.00 net. *Sold by Subscription.*

An American Text-Book *of* Legal Medicine *and* Toxicology.

Edited by Frederick Peterson, M. D., Chief of Clinic, Nervous Department, College of Physicians and Surgeons, New York; and Walter S. Haines, M. D., Professor of Chemistry, Pharmacy, and Toxicology, Rush Medical College, Chicago. *In Preparation.*

An American Text-Book *of* Obstetrics.

Edited by Richard C. Norris, M. D.; Art Editor, Robert L. Dickinson, M. D. Handsome imperial octavo volume of 1014 pages; nearly 900 beautiful colored and half-tone illustrations. Cloth, $7.00 net; Sheep or Half Morocco, $8.00 net. *Sold by Subscription.*

An American Text-Book *of* Pathology.

Edited by Ludwig Hektoen, M. D., Professor of Pathology in Rush Medical College, Chicago; and David Riesman, M. D., Demonstrator of Pathologic Histology in the University of Pennsylvania. *In Press, Ready Shortly.*

An American Text-Book *of* Physiology. Second Edition, Revised, in Two Volumes.

Edited by William H. Howell, Ph. D., M. D., Professor of Physiology, Johns Hopkins University, Baltimore, Md. Two royal octavo volumes of about 600 pages each. Fully illustrated. Per volume: Cloth, $3.00 net; Sheep or Half Morocco, $3.75 net.

An American Text-Book *of* Surgery. Third Edition.

Edited by William W. Keen, M. D., LL. D., F. R. C. S. (Hon.); and J. William White, M. D., Ph. D. Handsome octavo volume of 1230 pages; 496 wood-cuts and 37 colored and half-tone plates. Thoroughly revised and enlarged, with a section devoted to "The Use of the Röntgen Rays in Surgery." Cloth, $7.00 net; Sheep or Half Morocco,

THE NEW STANDARD **THE NEW STANDARD**

he American Illustrated Medical Dictionary.

For Practitioners and Students. A Complete Dictionary of the Terms used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, and the kindred branches, including much collateral information of an encyclopedic character, together with new and elaborate tables of Arteries, Muscles, Nerves, Veins, etc.; of Bacilli, Bacteria, Micrococci, Streptococci; Eponymic Tables of Diseases, Operations, Signs and Symptoms, Stains, Tests, Methods of Treatment, etc., etc. By W. A. NEWMAN DORLAND, A. M., M. D., Editor of the "American Pocket Medical Dictionary." Handsome large octavo, nearly 800 pages, bound in full flexible leather. Price, $4.50 net; with thumb index, $5.00 net.

ves a Maximum Amount of Matter in a Minimum Space and at the Lowest Possible Cost.

This is an entirely new and unique work, intended to meet the need of practitioners and dents for a complete up-to-date dictionary of moderate price. It contains more than twice material in the ordinary students' dictionary, and yet, by the use of a clear, condensed e and thin paper of the finest quality, it forms an extremely handy volume only 1¾ inches thickness. It is a beautiful specimen of the bookmaker's art. It is bound in full flexible ther, and is just the kind of book that a man will want to keep on his desk for constant erence. The book makes a special feature of the newer words, and defines hundreds of portant terms not to be found in any other dictionary. It is especially full in the matter tables, containing more than a hundred of great practical value. An important feature the book is its handsome illustrations and colored plates drawn especially for the work, luding new colored plates of Arteries, Nerves, Veins, Bacteria, Blood, etc.—twenty-four in . This new work has been aptly termed by a competent critic "The New Standard."

he American Pocket Medical Dictionary. **Third Edition, Revised.**

Edited by W. A. NEWMAN DORLAND, M. D., Assistant Obstetrician to the Hospital of the University of Pennsylvania; Fellow of the American Academy of Medicine. Containing the pronunciation and definition of the principal words used in medicine and kindred sciences, with 64 extensive tables. Handsomely bound in flexible leather, with gold edges. Price $1.00 net; with thumb index, $1.25 net.

he American Year-Book of Medicine and Surgery.

A Yearly Digest of Scientific Progress and Authoritative Opinion in all branches of Medicine and Surgery, drawn from journals, monographs, and text-books of the leading American and Foreign authors and investigators. Arranged with critical editorial comments, by eminent American specialists, under the editorial charge of GEORGE M. GOULD, M. D. Year-Book of 1901 in two volumes—Vol. I. including *General Medicine;* Vol. II., *General Surgery.* Per volume: Cloth, $3.00 net; Half Morocco, $3.75 net. *Sold by Subscription.*

bbott on Transmissible Diseases.

The Hygiene of Transmissible Diseases: their Causation, Modes of Dissemination, and Methods of Prevention. By A. C. ABBOTT, M. D., Professor of Hygiene and Bacteriology, University of Pennsylvania. Octavo, 311 pages with numerous illustrations. Cloth, $2.00 net.

Anders' Practice *of* Medicine. Fourth Revised Edition.

A Text-Book of the Practice of Medicine. By James M. Anders, M. D., Ph. D., LL. D., Professor of the Practice of Medicine and of Clinical Medicine, Medico-Chirurgical College, Philadelphia. Handsome octavo volume of 1292 pages, fully illustrated. Cloth, $5.50 net; Sheep or Half Morocco, $6.50 net.

Bastin's Botany.

Laboratory Exercises in Botany. By Edson S. Bastin, M. A., late Professor of Materia Medica and Botany, Philadelphia College of Pharmacy. Octavo, 536 pages, with 87 plates. Cloth, $2.00 net.

Beck on Fractures.

Fractures. By Carl Beck, M. D., Surgeon to St. Mark's Hospital and the New York German Poliklinik, etc. With an appendix on the Practical Use of the Röntgen Rays. 335 pages, 170 illustrations. Cloth, $3.50 net.

Beck's Surgical Asepsis.

A Manual of Surgical Asepsis. By Carl Beck, M. D., Surgeon to St. Mark's Hospital and the New York German Poliklinik, etc. 306 pages; 65 text-illustrations and 12 full-page plates. Cloth, $1.25 net.

Boislinière's Obstetric Accidents, Emergencies, *and* Operations.

Obstetric Accidents, Emergencies, and Operations. By L. Ch. Boislinière, M. D., late Emeritus Professor of Obstetrics, St. Louis Medical College. 381 pages, handsomely illustrated. Cloth, $2.00 net.

Böhm, Davidoff, *and* Huber's Histology.

A Text-Book of Human Histology. Including Microscopic Technic. By Dr. A. A. Böhm and Dr. M. von Davidoff, of Munich, and G. Carl Huber, M. D., Junior Professor of Anatomy and Director of Histological Laboratory, University of Michigan. Handsome octavo of 503 pages, with 351 beautiful original illustrations. Cloth, $3.50 net.

Butler's Materia Medica, Therapeutics, *and* Pharmacology. Third Edition, Revised.

A Text-Book of Materia Medica, Therapeutics, and Pharmacology. By George F. Butler, Ph. G., M. D., Professor of Materia Medica and of Clinical Medicine, College of Physicians and Surgeons, Chicago. Octavo, 874 pages, illustrated. Cloth, $4.00 net; Sheep or Half Morocco, $5.00 net.

Cerna on *the* Newer Remedies. Second Edition, Revised.

Notes on the Newer Remedies, their Therapeutic Applications and Modes of Administration. By David Cerna, M. D., Ph. D., Demonstrator of Physiology, Medical Department, University of Texas. Rewritten and greatly enlarged. Post-octavo, 253 pages. Cloth, $1.00 net.

Chapin on Insanity.

A Compendium of Insanity. By JOHN B. CHAPIN, M. D., LL. D. Physician-in-Chief, Pennsylvania Hospital for the Insane: Honorar Member of the Medico-Psychological Society of Great Britain, of th Society of Mental Medicine of Belgium, etc. 12mo, 234 pages, illustrated. Cloth, $1.25 net.

Chapman's Medical Jurisprudence *and* Toxicology

Second Edition, Revised.

Medical Jurisprudence and Toxicology. By HENRY C. CHAPMAN M. D., Professor of Institutes of Medicine and Medical Jurisprudence Jefferson Medical College of Philadelphia. 254 pages, with 55 illustrations and 3 full-page plates in colors. Cloth, $1.50 net.

Church *and* Peterson's Nervous *and* Mental Diseases

Second Edition.

Nervous and Mental Diseases. By ARCHIBALD CHURCH, M. D., Professor of Nervous and Mental Diseases, and Head of the Neurologica Department, Northwestern University Medical School, Chicago; an FREDERICK PETERSON, M. D., Chief of Clinic, Nervous Department College of Physicians and Surgeons, New York. Handsome octav volume of 843 pages, profusely illustrated. Cloth, $5.00 net; Sheep o Half Morocco, $6.00 net.

Clarkson's Histology.

A Text-Book of Histology, Descriptive and Practical. By ARTHU CLARKSON, M. B., C. M. Edin., formerly Demonstrator of Physiolog in the Owen's College, Manchester; late Demonstrator of Physiolog in Yorkshire College, Leeds. Large octavo, 554 pages, 22 engraving and 174 beautifully colored original illustrations. Cloth, $4.00 net.

Corwin's Physical Diagnosis. **Third Edition, Revised.**

Essentials of Physical Diagnosis of the Thorax. By ARTHUR M CORWIN, A. M., M. D., Instructor in Physical Diagnosis in Rus Medical College, Chicago. 219 pages, illustrated. Cloth, $1.25 net.

Crookshank's Bacteriology. **Fourth Edition, Revised.**

A Text-Book of Bacteriology. By EDGAR M. CROOKSHANK, M. B. Professor of Comparative Pathology and Bacteriology, King's College London. Octavo, 700 pages, [illegible] engravings, and 22 original colore plates. Cloth, $6.50 net; Half Morocco, $7.50 net.

DaCosta's Surgery. **Third Edition, Revised.**

Modern Surgery, General and Operative. By JOHN CHALMERS D COSTA, M. D., Professor of Principles of Surgery and Clinical Surgery Jefferson Medical College, Philadelphia; Surgeon to the Philadelphi Hospital, etc. Handsome octavo volume of 1117 pages, profusel illustrated. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

Enlarged by over 200 Pages, with more than 100 New Illustrations.

Davis's Obstetric Nursing.

Obstetric and Gynecologic Nursing. By EDWARD P. DAVIS, A. M., M. D., Professor of Obstetrics in Jefferson Medical College and the Philadelphia Polyclinic; Obstetrician and Gynecologist to the Philadelphia Hospital. 12mo volume of 400 pages, fully illustrated. *Ready Shortly.*

DeSchweinitz on Diseases *of the* Eye. Third Edition, Revised.

Diseases of the Eye. A Handbook of Ophthalmic Practice. By G. E. DE SCHWEINITZ, M. D., Professor of Ophthalmology, Jefferson Medical College, Philadelphia, etc. Handsome royal octavo volume of 696 pages; 256 fine illustrations and 2 chromo-lithographic plates. Cloth, $4.00 net; Sheep or Half Morocco, $5.00 net.

Dorland's Dictionaries.

[See *American Illustrated Medical Dictionary* and *American Pocket Medical Dictionary* on page 3.]

Dorland's Obstetrics.

A Manual of Obstetrics. By W. A. NEWMAN DORLAND, M. D., Assistant Demonstrator of Obstetrics, University of Pennsylvania; Associate in Gynecology, Philadelphia Polyclinic. 760 pages; 163 illustrations in the text and 6 full-page plates. Cloth, $2.50 net.

Eichhorst's Practice *of* Medicine.

A Text Book of the Practice of Medicine. By DR. HERMAN EICHHORST, Professor of Special Pathology and Therapeutics and Director of the Medical Clinic, University of Zurich. Translated and edited by AUGUSTUS A. ESHNER, M. D., Professor of Clinical Medicine, Philadelphia Polyclinic. *In Press. Ready Soon.*

Friedrich *and* Curtis on *the* Nose, Throat, *and* Ear.

Rhinology, Laryngology, and Otology, and Their Significance in General Medicine. By DR. E. P. FRIEDRICH, of Leipzig. Edited by H HOLBROOK CURTIS, M. D., Consulting Surgeon to the New York Nose and Throat Hospital. Octavo, 348 pages. Cloth, $2.50 net.

Frothingham's Guide for *the* Bacteriologist.

Laboratory Guide for the Bacteriologist. By LANGDON FROTHINGHAM, M. D. V., Assistant in Bacteriology and Veterinary Science, Sheffield Scientific School, Yale University. Illustrated. Cloth, 75 cts. net.

Garrigues' Diseases *of* Women. Third Edition, Revised.

Gould *and* Pyle's Curiosities *of* Medicine.

Anomalies and Curiosities of Medicine. By George M. Gould, M. D. and Walter L. Pyle, M. D. An encyclopedic collection of rare an extraordinary cases and of the most striking instances of abnormality i all branches of Medicine and Surgery, derived from an exhaustiv research of medical literature from its origin to the present day abstracted, classified, annotated, and indexed. Handsome octav volume of 968 pages; 295 engravings and 12 full-page plates. Popula Edition. Cloth, $3.00 net; Sheep or Half Morocco, $4.00 net.

Grafstrom's Mechano-Therapy.

A Text-Book of Mechano-Therapy (Massage and Medical Gymnastics) By Axel V. Grafstrom, B. Sc., M. D., late House Physician, City Hospital, Blackwell's Island, New York. 12mo, 139 pages, illustrated Cloth, $1.00 net.

Griffith on *the* Baby. Second Edition, Revised.

The Care of the Baby. By J. P. Crozer Griffith, M. D., Clinica Professor of Diseases of Children, University of Pennsylvania; Physician to the Children's Hospital, Philadelphia, etc. 12mo, 404 pages 67 illustrations and 5 plates. Cloth, $1.50 net.

Griffith's Weight Chart.

Infant's Weight Chart. Designed by J. P. Crozer Griffith, M. D. Clinical Professor of Diseases of Children, University of Pennsylvania 25 charts in each pad. Per pad, 50 cts. net.

Hart's Diet in Sickness *and* in Health.

Diet in Sickness and Health. By Mrs. Ernest Hart, formerly Studen of the Faculty of Medicine of Paris and of the London School of Medicine for Women; with an Introduction by Sir Henry Thompson F. R. C. S., M. D., London. 220 pages. Cloth, $1.50 net.

Haynes' Anatomy.

A Manual of Anatomy. By Irving S. Haynes, M. D., Professor of Practical Anatomy in Cornell University Medical College. 680 pages 42 diagrams and 134 full-page half-tone illustrations from original photographs of the author's dissections. Cloth, $2.50 net.

Heisler's Embryology.

A Text-Book of Embryology. By John C. Heisler, M. D., Professo of Anatomy, Medico-Chirurgical College, Philadelphia. Octavo volum of 405 pages, handsomely illustrated. Cloth, $2.50 net.

Hirst's Obstetrics. Second Edition.

A Text-Book of Obstetrics. By Barton Cooke Hirst, M. D., Professo of Obstetrics, University of Pennsylvania. Handsome octavo volum of 848 pages; 618 illustrations and 7 colored plates. Cloth, $5.00 net;

Hyde *and* Montgomery on Syphilis *and the* Venereal Diseases. Second Edition, Revised and Greatly Enlarged.

Syphilis and the Venereal Diseases. By JAMES NEVINS HYDE, M. D., Professor of Skin and Venereal Diseases, and FRANK H. MONTGOMERY, M. D., Associate Professor of Skin, Genito-Urinary, and Venereal Diseases in Rush Medical College, Chicago, Ill. Octavo, 594 pages, profusely illustrated. Cloth, $4.00 net.

The International Text-Book of Surgery. In Two Volumes.

By American and British Authors. Edited by J. COLLINS WARREN, M. D., LL. D., F. R. C. S. (Hon.), Professor of Surgery, Harvard Medical School, Boston; and A. PEARCE GOULD, M. S., F. R. C. S., Lecturer on Practical Surgery and Teacher of Operative Surgery, Middlesex Hospital Medical School, London, Eng. Vol. I. *General Surgery.*—Handsome octavo, 947 pages, with 458 beautiful illustrations and 9 lithographic plates. Vol. II. *Special or Regional Surgery.*—Handsome octavo, 1072 pages, with 471 beautiful illustrations and 8 lithographic plates. *Sold by Subscription.* Prices per volume: Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

"It is the most valuable work on the subject that has appeared in some years. The clinician and the pathologist have joined hands in its production, and the result must be a satisfaction to the editors as it is a gratification to the conscientious reader."—*Annals of Surgery.*

"This is a work which comes to us on its own intrinsic merits. Of the latter it has very many. The arrangement of subjects is excellent, and their treatment by the different authors is equally so. What is especially to be recommended is the painstaking endeavor of each writer to make his subject clear and to the point. To this end particularly is the technique of operations lucidly described in all necessary detail. And withal the work is up to date in a very remarkable degree, many of the latest operations in the different regional parts of the body being given in full details. There is not a chapter in the work from which the reader may not learn something new."—*Medical Record*, New York.

Jackson's Diseases *of the* Eye.

A Manual of Diseases of the Eye. By EDWARD JACKSON, A. M., M. D., Emeritus Professor of Diseases of the Eye, Philadelphia Polyclinic and College for Graduates in Medicine. 12mo volume of 535 pages, with 178 illustrations, mostly from drawings by the author. Cloth, $2.50 net.

Keating's Life Insurance.

How to Examine for Life Insurance. By JOHN M. KEATING, M. D., Fellow of the College of Physicians of Philadelphia; Ex-President of the Association of Life Insurance Medical Directors. Royal octavo, 211 pages. With numerous illustrations. Cloth, $2.00 net.

Keen on *the* Surgery *of* Typhoid Fever.

The Surgical Complications and Sequels of Typhoid Fever. By WM. W. KEEN, M. D., LL. D., F. R. C. S. (Hon.), Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College, Philadelphia, etc. Octavo volume of 386 pages, illustrated. Cloth, $3.00 net.

Keen's Operation Blank. Second Edition, Revised Form.

An Operation Blank, with Lists of Instruments, etc., Required in Various Operations. Prepared by W. W. KEEN, M. D., LL. D., F. R. C. S. (Hon.), Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College, Philadelphia. Price per pad, blanks for fifty operations, 50 cts. net.

Kyle on *the* Nose and Throat. **Second Edition.**

Diseases of the Nose and Throat. By D. BRADEN KYLE, M. D., Clir Professor of Laryngology and Rhinology, Jefferson Medical Coll Philadelphia. Octavo, 646 pages; over 150 illustrations and 6 li graphic plates. Cloth, $4.00 net; Sheep or Half Morocco, $5.00 r

Laine's Temperature Chart.

Temperature Chart. Prepared by D. T. LAINÉ, M. D. Size 8 x 1 inches. A conveniently arranged Chart for recording Temperat with columns for daily amounts of Urinary and Fecal Excretions, F Remarks, etc. On the back of each chart is given the Brand treatn of Typhoid Fever. Price, per pad of 25 charts, 50 cts. net.

Levy, Klemperer, *and* Eshner's Clinical Bacteriolo

The Elements of Clinical Bacteriology. By DR. ERNST LEVY, fessor in the University of Strasburg, and FELIX KLEMPERER, Pri docent in the University of Strasburg. Translated and edited AUGUSTUS A. ESHNER, M. D., Professor of Clinical Medicine, Phila phia Polyclinic. Octavo, 440 pages, fully illustrated. Cloth, $2.50

Lockwood's Practice *of* Medicine.

A Manual of the Practice of Medicine. By GEORGE ROE LOCKWC M. D., Professor of Practice in the Woman's Medical College of New York Infirmary, etc. 935 pages, with 75 illustrations in the t and 22 full-page plates. Cloth, $2.50 net.

Long's Syllabus *of* Gynecology.

A Syllabus of Gynecology, arranged in Conformity with "An Ameri Text-Book of Gynecology." By J. W. LONG, M. D., Professor of eases of Women and Children, Medical College of Virginia, etc. Cl interleaved, $1.00 net.

Macdonald's Surgical Diagnosis *and* Treatment.

Surgical Diagnosis and Treatment. By J. W. MACDONALD, M Edin., F. R. C. S. Edin., Professor of Practice of Surgery and Clin Surgery, Hamline University. Handsome octavo, 800 pages, fully il trated. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

Mallory *and* Wright's Pathological Technique.

Pathological Technique. A Practical Manual for Laboratory Worl Pathology, Bacteriology, and Morbid Anatomy, with chapters on P Mortem Technique and the Performance of Autopsies. By FRANK MALLORY, A. M., M. D., Assistant Professor of Pathology, Harv University Medical School, Boston; and JAMES H. WRIGHT, A. M. D., Instructor in Pathology, Harvard University Medical Sch Boston. Octavo, 396 pages, handsomely illustrated. Cloth, $2.50 r

McFarland's Pathogenic Bacteria. **Third Edition, increase size by over 100 Page**

Text-Book upon the Pathogenic Bacteria. By JOSEPH McFARLA M. D., Professor of Pathology and Bacteriology, Medico-Chirurg College of Philadelphia, etc. Octavo volume of 621 pages, finely il *trated.* *Cloth*, $3.25 net.

Meigs on Feeding in Infancy.

Feeding in Early Infancy. By ARTHUR V. MEIGS, M. D. Bound in limp cloth, flush edges, 25 cts. net.

Moore's Orthopedic Surgery.

A Manual of Orthopedic Surgery. By JAMES E. MOORE, M. D., Professor of Orthopedics and Adjunct Professor of Clinical Surgery, University of Minnesota, College of Medicine and Surgery. Octavo volume of 356 pages, handsomely illustrated. Cloth, $2.50 net.

Morten's Nurses' Dictionary.

Nurses' Dictionary of Medical Terms and Nursing Treatment.. Containing Definitions of the Principal Medical and Nursing Terms and Abbreviations; of the Instruments, Drugs, Diseases, Accidents, Treatments, Operations, Foods, Appliances, etc. encountered in the ward or in the sick-room. By HONNOR MORTEN, author of "How to Become a Nurse," etc. 16mo, 140 pages. Cloth, $1.00 net.

Nancrede's Anatomy *and* Dissection. **Fourth Edition.**

Essentials of Anatomy and Manual of Practical Dissection. By CHARLES B. NANCREDE, M. D., LL. D., Professor of Surgery and of Clinical Surgery, University of Michigan, Ann Arbor. Post-octavo, 500 pages, with full-page lithographic plates in colors and nearly 200 illustrations. Extra Cloth (or Oilcloth for dissection-room), $2.00 net.

Nancrede's Principles *of* Surgery.

Lectures on the Principles of Surgery. By CHAS. B. NANCREDE, M. D., LL. D., Professor of Surgery and of Clinical Surgery, University of Michigan, Ann Arbor. Octavo, 398 pages, illustrated. Cloth, $2.50 net.

Norris's Syllabus *of* Obstetrics. **Third Edition, Revised.**

Syllabus of Obstetrical Lectures in the Medical Department of the University of Pennsylvania. By RICHARD C. NORRIS, A. M., M. D., Instructor in Obstetrics and Lecturer on Clinical and Operative Obstetrics, University of Pennsylvania. Crown octavo, 222 pages. Cloth, interleaved for notes, $2.00 net.

Ogden on *the* Urine.

Clinical Examination of the Urine and Urinary Diagnosis. A Clinical Guide for the Use of Practitioners and Students of Medicine and Surgery. By J. BERGEN OGDEN, M. D., Instructor in Chemistry, Harvard Medical School. Handsome octavo, 416 pages, with 54 illustrations and a number of colored plates. Cloth, $3.00 net.

Penrose's Diseases *of* Women. **Third Edition, Revised.**

A Text-Book of Diseases of Women. By CHARLES B. PENROSE, M. D., PH. D., formerly Professor of Gynecology in the University of Pennsylvania. Octavo volume of 531 pages, handsomely illustrated. Cloth,

Pryor—Pelvic Inflammations.

The Treatment of Pelvic Inflammations through the Vagina. By W. R. PRYOR, M. D., Professor of Gynecology, New York Polyclinic. 12mo, 248 pages, handsomely illustrated. Cloth, $2.00 net.

Pye's Bandaging.

Elementary Bandaging and Surgical Dressing. With Directions concerning the Immediate Treatment of Cases of Emergency. By WALTER PYE, F. R. C. S., late Surgeon to St. Mary's Hospital, London. Small 12mo, over 80 illustrations. Cloth, flexible covers, 75 cts. net.

Pyle's Personal Hygiene.

A Manual of Personal Hygiene. Proper Living upon a Physiologic Basis. Edited by WALTER L. PYLE, M. D., Assistant Surgeon to the Wills Eye Hospital, Philadelphia. Octavo volume of 344 pages, fully illustrated. Cloth, $1.50 net.

Raymond's Physiology.

A Manual of Physiology. By JOSEPH H. RAYMOND, A. M., M. D., Professor of Physiology and Hygiene and Lecturer on Gynecology in the Long Island College Hospital. 382 pages, 102 illustrations, and 4 full-page colored plates. Cloth, $1.25 net.

Salinger *and* Kalteyer's Modern Medicine.

Modern Medicine. By JULIUS L. SALINGER, M. D., Demonstrator of Clinical Medicine, Jefferson Medical College; and F. J. KALTEYER, M. D., Assistant Demonstrator of Clinical Medicine, Jefferson Medical College. Handsome octavo, 801 pages, illustrated. Cloth, $4.00 net.

Saundby's Renal *and* Urinary Diseases.

Lectures on Renal and Urinary Diseases. By ROBERT SAUNDBY, M. D. Edin., Fellow of the Royal College of Physicians, London, and of the Royal Medico-Chirurgical Society; Professor of Medicine in Mason College, Birmingham, etc. Octavo, 434 pages, with numerous illustrations and 4 colored plates. Cloth, $2.50 net.

Saunders' Medical Hand-Atlases.

See pages 16 and 17.

Saunders' Pocket Medical Formulary. **Sixth Edition, Revised.**

By WILLIAM M. POWELL, M. D., author of "Essentials of Diseases of Children"; Member of Philadelphia Pathological Society. Containing 1844 formulæ from the best-known authorities. With an Appendix containing Posological Table, Formulæ and Doses for Hypodermic Medication, Poisons and their Antidotes, Diameters of the Female Pelvis and Fetal Head, Obstetrical Table, Diet List for Various Diseases, Materials and Drugs used in Antiseptic Surgery, Treatment of Asphyxia from Drowning, Surgical Remembrancer, Tables of Incompatibles, Eruptive Fevers, etc., etc. Handsomely bound in flexible morocco, with side index, wallet, and flap. $2.00 net.

Saunders' Question-Compends.

See page 15.

Scudder's Fractures. Second Edition, Revised.

The Treatment of Fractures. By Chas. L. Scudder, M. D., Assistant in Clinical and Operative Surgery, Harvard University Medical School. Octavo, 460 pages, with nearly 600 original illustrations. Polished Buckram, $4.50 net; Half Morocco, $5.50 net.

Senn's Genito-Urinary Tuberculosis.

Tuberculosis of the Genito-Urinary Organs, Male and Female. By Nicholas Senn, M. D., Ph. D., LL. D., Professor of the Practice of Surgery and of Clinical Surgery, Rush Medical College, Chicago. Handsome octavo volume of 320 pages, illustrated. Cloth, $3.00 net.

Senn's Practical Surgery.

Practical Surgery. By Nicholas Senn, M. D., Ph. D., LL. D., Professor of the Practice of Surgery and of Clinical Surgery, Rush Medical College, Chicago. Handsome octavo volume of over 1000 pages, profusely illustrated. *In Press.*

Senn's Syllabus *of* Surgery.

A Syllabus of Lectures on the Practice of Surgery, arranged in conformity with "An American Text-Book of Surgery." By Nicholas Senn, M. D., Ph. D., LL. D., Professor of the Practice of Surgery and of Clinical Surgery, Rush Medical College, Chicago. Cloth, $1.50 net.

Senn's Tumors. Second Edition, Revised.

Pathology and Surgical Treatment of Tumors. By Nicholas Senn, M. D., Ph. D., LL. D., Professor of the Practice of Surgery and of Clinical Surgery, Rush Medical College, Chicago. Octavo volume of 718 pages, with 478 illustrations, including 12 full-page plates in colors. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

Starr's Diets for Infants *and* Children.

Diets for Infants and Children in Health and in Disease. By Louis Starr, M. D., Editor of "An American Text-Book of the Diseases of Children." 230 blanks (pocket-book size), perforated and neatly bound in flexible morocco. $1.25 net.

Stengel's Pathology. Third Edition, Thoroughly Revised.

A Text-Book of Pathology. By Alfred Stengel, M. D., Professor of Clinical Medicine, University of Pennsylvania; Visiting Physician to the Pennsylvania Hospital. Handsome octavo, 873 pages, nearly 400 illustrations, many of them in colors. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

Stengel *and* White on *the* Blood.

The Blood in its Clinical and Pathological Relations. By Alfred Stengel, M. D., Professor of Clinical Medicine, University of Pennsylvania; and C. Y. White, Jr., M. D., Instructor in Clinical Medicine, University of Pennsylvania. *In Press.*

Stevens' Materia Medica and Therapeutics. Second Editio Revised.

A Manual of Materia Medica and Therapeutics. By A. A. STEVEN A. M., M. D., Lecturer on Physical Diagnosis in the University o Pennsylvania. Post-octavo, 445 pages. Flexible Leather, $2.00 ne

Stevens' Practice of Medicine. Fifth Edition, Revised.

A Manual of the Practice of Medicine. By A. A. STEVENS, A. M. M. D., Lecturer on Physical Diagnosis in the University of Pennsyl vania. Specially intended for students preparing for graduation an hospital examinations. Post-octavo, 519 pages; illustrated. Flexibl Leather, $2.00 net.

Stewart's Physiology. Fourth Edition, Revised.

A Manual of Physiology, with Practical Exercises. For Students an Practitioners. By G. N. STEWART, M. A., M. D., D. Sc., Professor o Physiology in the Western Reserve University, Cleveland, Ohio. Octav volume of 894 pages; 336 illustrations and 5 colored plates. Cloth $3.75 net.

Stoney's Materia Medica for Nurses.

Materia Medica for Nurses. By EMILY A. M. STONEY, late Superintend ent of the Training-School for Nurses, Carney Hospital, South Boston Mass. Handsome octavo volume of 306 pages. Cloth, $1.50 net.

Stoney's Nursing. Second Edition, Revised.

Practical Points in Nursing. For Nurses in Private Practice. By EMIL A. M. STONEY, late Superintendent of the Training-School for Nurses Carney Hospital, South Boston, Mass. 456 pages, with 73 engraving and 8 colored and half-tone plates. Cloth, $1.75 net.

Stoney's Surgical Technic for Nurses.

Bacteriology and Surgical Technic for Nurses. By EMILY A. M. STONEY late Superintendent of the Training-School for Nurses, Carney Hospital South Boston, Mass. 12mo volume, fully illustrated. Cloth, $1.25 net

Thomas's Diet Lists. Second Edition, Revised.

Diet Lists and Sick-Room Dietary. By JEROME B. THOMAS, M. D., Visiting Physician to the Home for Friendless Women and Childre and to the Newsboys' Home; Assistant Visiting Physician to the King County Hospital. Cloth, $1.25 net. Send for sample sheet.

Thornton's Dose-Book and Prescription-Writing.

Dose-Book and Manual of Prescription-Writing. By E. Q. THORNTON, M. D., Demonstrator of Therapeutics, Jefferson Medical College, Phila delphia. 334 pages, illustrated. Cloth, $1.25 net.

Van Valzah and Nisbet's Diseases of the Stomach.

Diseases of the Stomach. By WILLIAM W. VAN VALZAH, M. D., Professor of General Medicine and Diseases of the Digestive System and the Blood, New York Polyclinic; and J. DOUGLAS NISBET, M. D., Adjunct Professor of General Medicine and Diseases of the Digestive System and the Blood, New York Polyclinic. Octavo volume of 674 pages, illustrated. Cloth, $3.50 net.

Vecki's Sexual Impotence.

The Pathology and Treatment of Sexual Impotence. By VICTOR G. VECKI, M. D. From the second German edition, revised and enlarged. Demi-octavo, 291 pages. Cloth, $2.00 net.

Vierordt's Medical Diagnosis. **Fourth Edition, Revised.**

Medical Diagnosis. By DR. OSWALD VIERORDT, Professor of Medicine, University of Heidelberg. Translated, with additions, from the fifth enlarged German edition, with the author's permission, by FRANCIS H. STUART, A. M., M. D. Handsome octavo volume, 603 pages; 194 wood-cuts, many of them in colors. Cloth, $4.00 net; Sheep or Half Morocco, $5.00 net.

Watson's Handbook for Nurses.

A Handbook for Nurses. By J. K. WATSON, M. D. Edin. American Edition, under supervision of A. A. STEVENS, A. M., M. D., Lecturer on Physical Diagnosis, University of Pennsylvania. 12mo, 413 pages, 73 illustrations. Cloth, $1.50 net.

Warren's Surgical Pathology. **Second Edition.**

Surgical Pathology and Therapeutics. By JOHN COLLINS WARREN, M. D., LL. D., F. R. C. S. (Hon.), Professor of Surgery, Harvard Medical School. Handsome octavo, 873 pages; 136 relief and lithographic illustrations, 33 in colors. With an Appendix on Scientific Aids to Surgical Diagnosis, and a series of articles on Regional Bacteriology. Cloth, $5.00 net; Sheep or Half Morocco, $6.00 net.

SAUNDERS'

QUESTION-COMPENDS.

ARRANGED IN QUESTION AND ANSWER FORM.

The Most Complete and Best Illustrated Series of Compends Ever Issued.

NOW THE STANDARD AUTHORITIES IN MEDICAL LITERATURE

WITH

Students and Practitioners in every City of the United States and Canada.

Since the issue of the first volume of the Saunders Question-Compends,

OVER 175,000 COPIES

of these unrivalled publications have been sold. This enormous sale is indisputable evidence of the value of these self-helps to students and physicians.

SEE NEXT PAGE FOR LIST.

SAUNDERS'
QUESTION-COMPEND SERIE

Price, Cloth, $1.00 net per copy, except when otherwise noted.

"Where the work of preparing students' manuals is to end we cannot say, but the Saunders Seri in our opinion, bears off the palm at present."—*New York Medical Record.*

1. **Essentials of Physiology.** A new work in preparation.
2. **Essentials of Surgery.** By EDWARD MARTIN, M. D. Seventh edition, revised, an Appendix and a chapter on Appendicitis.
3. **Essentials of Anatomy.** By CHARLES B. NANCREDE, M. D. Sixth edition, oughly revised and enlarged.
4. **Essentials of Medical Chemistry, Organic and Inorganic.** By LAWRENCE W M. D. Fifth edition, revised.
5. **Essentials of Obstetrics.** By W. EASTERLY ASHTON, M. D. Fourth edition, re and enlarged.
6. **Essentials of Pathology and Morbid Anatomy.** By F. J. KALTEYER, M. D. *preparation.*
7. **Essentials of Materia Medica, Therapeutics, and Prescription-Writing.** By H MORRIS, M. D. Fifth edition, revised.

8, 9. **Essentials of Practice of Medicine.** By HENRY MORRIS, M. D. An App on URINE EXAMINATION. By LAWRENCE WOLFF, M. D. Third edition, enl by some 300 Essential Formulæ, selected from eminent authorities, by W POWELL, M. D. (Double number, $1.50 net.)

10. **Essentials of Gynecology.** By EDWIN B. CRAGIN, M. D. Fourth edition, re
11. **Essentials of Diseases of the Skin.** By HENRY W. STELWAGON, M. D. F edition, revised and enlarged.
12. **Essentials of Minor Surgery, Bandaging, and Venereal Diseases.** By ED MARTIN, M. D. Second edition, revised and enlarged.
13. **Essentials of Legal Medicine, Toxicology, and Hygiene.** This volume present out of print.
14. **Essentials of Diseases of the Eye, Nose, and Throat.** By EDWARD JAC M. D., and E. B. GLEASON, M. D. Second edition, revised.
15. **Essentials of Diseases of Children.** By WILLIAM M. POWELL, M. D. S edition.
16. **Essentials of Examination of Urine.** By LAWRENCE WOLFF, M. D. C "VOGEL SCALE." (75 cents net.)
17. **Essentials of Diagnosis.** By S. SOLIS-COHEN, M. D., and A. A. ESHNER, Second edition, thoroughly revised.
18. **Essentials of Practice of Pharmacy.** By LUCIUS E. SAYRE. Second ed revised and enlarged.

20. **Essentials of Bacteriology.** By M. V. BALL, M. D. Fourth edition, revised.
21. **Essentials of Nervous Diseases and Insanity.** By JOHN C. SHAW, M. D. edition, revised.
22. **Essentials of Medical Physics.** By FRED J. BROCKWAY, M. D. Second e revised.
23. **Essentials of Medical Electricity.** By DAVID D. STEWART, M. D., and ED S. LAWRANCE, M. D.
24. **Essentials of Diseases of the Ear.** By E. B. GLEASON, M. D. Second ed revised and greatly enlarged.

A New Volume.

25. **Essentials of Histology.** By LOUIS LEROY, M. D. With 73 original illustratic

Saunders' Medical Hand-Atlases.

VOLUMES NOW READY.

Atlas and Epitome of Internal Medicine and Clinical Diagnosis.

By DR. CHR. JAKOB, of Erlangen. Edited by AUGUSTUS A. ESHNER, M. D., Professor of Clinical Medicine, Philadelphia Polyclinic. With 179 colored figures on 68 plates, 64 text-illustrations, 259 pages of text. Cloth, $3.00 net.

Atlas of Legal Medicine.

By DR. E. R. VON HOFMANN, of Vienna. Edited by FREDERICK PETERSON, M. D., Chief of Clinic, Nervous Department, College of Physicians and Surgeons, New York. With 120 colored figures on 56 plates and 193 beautiful half-tone illustrations. Cloth, $3.50 net.

Atlas and Epitome of Diseases of the Larynx.

By DR. L. GRUNWALD, of Munich. Edited by CHARLES P. GRAYSON, M. D., Physician-in-Charge, Throat and Nose Department, Hospital of the University of Pennsylvania. With 107 colored figures on 44 plates, 25 text-illustrations, and 103 pages of text. Cloth, $2.50 net.

Atlas and Epitome of Operative Surgery.

By DR. O. ZUCKERKANDL, of Vienna. Edited by J. CHALMERS DACOSTA, M. D., Professor of Principles of Surgery and Clinical Surgery, Jefferson Medical College, Philadelphia. With 24 colored plates, 217 text-illustrations, and 395 pages of text. Cloth, $3.00 net.

Atlas and Epitome of Syphilis and the Venereal Diseases.

By PROF. DR. FRANZ MRACEK, of Vienna. Edited by L. BOLTON BANGS, M. D., Professor of Genito-Urinary Surgery, University and Bellevue Hospital Medical College, New York. With 71 colored plates, 16 illustrations, and 122 pages of text. Cloth, $3.50 net.

Atlas and Epitome of External Diseases of the Eye.

By DR. O. HAAB, of Zurich. Edited by G. E. DE SCHWEINITZ, M. D., Professor of Ophthalmology, Jefferson Medical College, Philadelphia. With 76 colored illustrations on 40 plates and 228 pages of text. Cloth, $3.00 net.

Atlas and Epitome of Skin Diseases.

By PROF. DR. FRANZ MRACEK, of Vienna. Edited by HENRY W. STELWAGON, M. D., Clinical Professor of Dermatology, Jefferson Medical College, Philadelphia. With 63 colored plates, 39 half-tone illustrations, and 200 pages of text. Cloth, $3.50 net.

Atlas and Epitome of Special Pathological Histology.

By DR. H. DURCK, of Munich. Edited by LUDWIG HEKTOEN M. D., Professor of Pathology, Rush Medical College, Chicago. In Two Parts. Part I. *Ready*, including Circulatory, Respiratory, and Gastro-intestinal Tract, 120 colored figures on 62 plates, 158 pages of text. Part II. *Ready Shortly*. Price of Part I., $3.00 net.

Saunders' Medical Hand-Atlases

VOLUMES JUST ISSUED.

Atlas and Epitome of Diseases Caused by Acciden

By DR. ED. GOLEBIEWSKI, of Berlin. Translated and edited with ad
tions by PEARCE BAILEY, M. D., Attending Physician to the Departm
of Corrections and to the Almshouse and Incurable Hospitals, N
York. With 40 colored plates, 143 text-illustrations, and 600 pa
of text. Cloth, $4.00 net.

Atlas and Epitome of Gynecology.

By DR. O. SHAEFFER, of Heidelberg. *From the Second Revised G*
man Edition. Edited by RICHARD C. NORRIS, A. M., M. D., Gy
cologist to the Methodist Episcopal and the Philadelphia Hospita
Surgeon-in-Charge of Preston Retreat, Philadelphia. With 90 colo
plates, 65 text-illustrations, and 308 pages of text. Cloth, $3.50 n

Atlas and Epitome of the Nervous System and i Diseases.

By PROFESSOR DR. CHR. JAKOB, of Erlangen. *From the Second*
vised German Edition. Edited by EDWARD D. FISHER, M. D., P
fessor of Diseases of the Nervous System, University and Belle
Hospital Medical College, New York. With 83 plates and a copi
text. Cloth.

Atlas and Epitome of Labor and Operative O stetrics.

By DR. O. SHAEFFER, of Heidelberg. *From the Fifth Revised Germ*
Edition. Edited by J. CLIFTON EDGAR, M. D., Professor of Obstetr
and Clinical Midwifery, Cornell University Medical School. With
colored illustrations. Cloth.

Atlas and Epitome of Obstetrical Diagnosis a Treatment.

By DR. O. SHAEFFER, of Heidelberg. *From the Second Revised Germ*
Edition. Edited by J. CLIFTON EDGAR, M. D., Professor of Obstetr
and Clinical Midwifery, Cornell University Medical School. 72 colo
plates, numerous text-illustrations, and copious text. Cloth.

IN PRESS FOR EARLY PUBLICATION.

Atlas and Epitome of Ophthalmoscopy and Op thalmoscopic Diagnosis.

By DR. O. HAAB, of Zürich. *From the Third Revised and Enlar*
German Edition. Edited by G. E. DE SCHWEINITZ, M. D., Profes
of Ophthalmology, Jefferson Medical College, Philadelphia. With
colored figures and 82 pages of text.

CLASSIFIED LIST
OF THE
MEDICAL PUBLICATIONS
OF
W. B. SAUNDERS & COMPANY

ANATOMY, EMBRYOLOGY, HISTOLOGY.

Böhm, Davidoff, and Huber—A Text-Book of Histology, ... 4
Clarkson—A Text-Book of Histology, . . 5
Haynes—A Manual of Anatomy, ... 7
Heisler—A Text-Book of Embryology, . . 7
Leroy—Essentials of Histology, ... 15
Nancrede—Essentials of Anatomy, ... 15
Nancrede—Essentials of Anatomy and Manual of Practical Dissection, ... 10

BACTERIOLOGY.

Ball—Essentials of Bacteriology, ... 15
Crookshank—A Text-Book of Bacteriology, ... 5
Frothingham—Laboratory Guide, ... 6
Levy and Klemperer's Clinical Bacteriology, ... 9
Mallory and Wright—Pathological Technique, ... 9
McFarland—Pathogenic Bacteria, ... 9

CHARTS, DIET-LISTS, ETC.

Griffith—Infant's Weight Chart, ... 7
Hart—Diet in Sickness and in Health, . . 7
Keen—Operation Blank, ... 8
Laine—Temperature Chart, ... 9
Meigs—Feeding in Early Infancy, ... 10
Starr—Diets for Infants and Children, . . 12
Thomas—Diet-Lists, ... 13

CHEMISTRY AND PHYSICS.

Brockway—Essentials of Medical Physics, 15
Wolff—Essentials of Medical Chemistry, . 15

CHILDREN.

An American Text-Book of Diseases of Children, ... 1
Griffith—Care of the Baby, ... 7
Griffith—Infant's Weight Chart, ... 7
Meigs—Feeding in Early Infancy, ... 10
Powell—Essentials of Diseases of Children, 15
Starr—Diets for Infants and Children, . . 12

DIAGNOSIS.

Cohen and Eshner—Essentials of Diagnosis, ... 15
Corwin—Physical Diagnosis, ... 5
Macdonald—Surgical Diagnosis and Treatment, ... 9
Vierordt—Medical Diagnosis, ... 14

DICTIONARIES.

The American Illustrated Medical Dictionary, ... 3
The American Pocket Medical Dictionary, 3
Morten—Nurses' Dictionary, ... 10

EYE, EAR, NOSE, AND THROAT.

An American Text-Book of Diseases of the Eye, Ear, Nose, and Throat, ... 1
De Schweinitz—Diseases of the Eye, . . 6
Friedrich and Curtis—Rhinology, Laryngology, and Otology, and their Significance in General Medicine, ... 6
Gleason—Essentials of Diseases of the Ear, 15
Grünwald and Grayson—Atlas of Diseases of the Larynx, ... 16
Haab and De Schweinitz—Atlas of External Diseases of the Eye, ... 16
Jackson—Manual of Diseases of the Eye, 8
Jackson and Gleason—Essentials of Diseases of the Eye, Nose, and Throat, . . 15
Kyle—Diseases of the Nose and Throat, . 9

GENITO-URINARY.

An American Text-Book of Genito-Urinary and Skin Diseases, ... 2
Hyde and Montgomery—Syphilis and the Venereal Diseases, ... 8
Martin—Essentials of Minor Surgery, Bandaging, and Venereal Diseases, . . . 15
Mracek and Bangs—Atlas of Syphilis and the Venereal Diseases, ... 16
Saundby—Renal and Urinary Diseases, . . 11
Senn—Genito-Urinary Tuberculosis, . . . 12
Vecki—Sexual Impotence, ... 14

GYNECOLOGY.

American Text-Book of Gynecology, . . 2
Cragin—Essentials of Gynecology, ... 15
Garrigues—Diseases of Women, ... 6
Long—Syllabus of Gynecology, ... 9
Penrose—Diseases of Women, ... 10
Pryor—Pelvic Inflammations, ... 11
Schaeffer and Norris—Atlas of Gynecology, ... 17

MATERIA MEDICA, PHARMACOLOGY, AND THERAPEUTICS.

An American Text-Book of Applied Therapeutics, ... 1
Butler—Text-Book of Materia Medica, Therapeutics, and Pharmacology, . . . 4
Cerna—Notes on the Newer Remedies, . . 4
Morris—Essentials of Materia Medica and Therapeutics, ... 15
Saunders' Pocket Medical Formulary, . . 11
Sayre—Essentials of Pharmacy, ... 15
Stevens—Manual of Therapeutics, ... 13
Stoney—Materia Medica for Nurses, . . . 13
Thornton—Dose-Book and Manual of Prescription-Writing, ... 13

MEDICAL PUBLICATIONS OF W. B. SAUNDERS & CO.

MEDICAL JURISPRUDENCE AND TOXICOLOGY.

Chapman—Medical Jurisprudence and Toxicology, 5
Golebiewski and Bailey—Atlas of Diseases Caused by Accidents, 17
Hofmann and Peterson—Atlas of Legal Medicine, 16

NERVOUS AND MENTAL DISEASES, ETC.

Chapin—Compendium of Insanity, 5
Church and Peterson—Nervous and Mental Diseases, 5
Shaw—Essentials of Nervous Diseases and Insanity, 15

NURSING.

Davis—Obstetric and Gynecologic Nursing, 6
Griffith—The Care of the Baby, 7
Hart—Diet in Sickness and in Health, 7
Meigs—Feeding in Early Infancy, 10
Morten—Nurses' Dictionary, 10
Stoney—Materia Medica for Nurses, 13
Stoney—Practical Points in Nursing, 13
Stoney—Surgical Technic for Nurses, 13
Watson—Handbook for Nurses, 14

OBSTETRICS.

An American Text-Book of Obstetrics, 2
Ashton—Essentials of Obstetrics, 15
Boislinière—Obstetric Accidents, 4
Dorland—Manual of Obstetrics, 6
Hirst—Text-Book of Obstetrics, 7
Norris—Syllabus of Obstetrics, 10
Schaeffer and Edgar—Atlas of Obstetrical Diagnosis and Treatment, 17

PATHOLOGY.

An American Text-Book of Pathology, 2
Dürck and Hektoen—Atlas of Pathologic Histology, 16
Kalteyer—Essentials of Pathology, 15
Mallory and Wright—Pathological Technique, 9
Senn—Pathology and Surgical Treatment of Tumors, 12
Stengel—Text-Book of Pathology, 12
Warren—Surgical Pathology and Therapeutics, 14

PHYSIOLOGY.

An American Text-Book of Physiology, 2
—Essentials of Physiology, 15
Raymond—Manual of Physiology, 11
Stewart—Manual of Physiology, 13

PRACTICE OF MEDICINE.

An American Year-Book of Medicine and Surgery, 3
Anders—Text-Book of the Practice of Medicine, 4
Eichhorst—Practice of Medicine, 6
Lockwood—Manual of the Practice of Medicine, 9
Morris—Essentials of Practice of Medicine, 15
Salinger and Kalteyer—Modern Medicine, 11
Stevens—Manual of Practice of Medicine, 13

SKIN AND VENEREAL.

An American Text-Book of Genito-Urinary and Skin Diseases,
Hyde and Montgomery—Syphilis and the Venereal Diseases,
Martin—Essentials of Minor Surgery, Bandaging, and Venereal Diseases,
Mracek and Stelwagon—Atlas of Diseases of the Skin,
Stelwagon—Essentials of Diseases of the Skin,

SURGERY.

An American Text-Book of Surgery,
An American Year-Book of Medicine and Surgery,
Beck—Fractures,
Beck—Manual of Surgical Asepsis,
Da Costa—Manual of Surgery,
International Text-Book of Surgery,
Keen—Operation Blank,
Keen—The Surgical Complications and Sequels of Typhoid Fever,
Macdonald—Surgical Diagnosis and Treatment,
Martin—Essentials of Minor Surgery, Bandaging, and Venereal Diseases,
Martin—Essentials of Surgery,
Moore—Orthopedic Surgery,
Nancrede—Principles of Surgery,
Pye—Bandaging and Surgical Dressing,
Scudder—Treatment of Fractures,
Senn—Genito-Urinary Tuberculosis,
Senn—Practical Surgery,
Senn—Syllabus of Surgery,
Senn—Pathology and Surgical Treatment of Tumors,
Warren—Surgical Pathology and Therapeutics,
Zuckerkandl and Da Costa—Atlas of Operative Surgery,

URINE AND URINARY DISEAS

Ogden—Clinical Examination of the Urine,
Saundby—Renal and Urinary Diseases,
Wolff—Essentials of Examination of Urine,

MISCELLANEOUS.

Abbott—Hygiene of Transmissible Diseases,
Bastin—Laboratory Exercises in Botany,
Golebiewski and Bailey—Atlas of Diseases Caused by Accidents,
Gould and Pyle—Anomalies and Curiosities of Medicine,
Grafstrom—Massage,
Keating—How to Examine for Life Insurance,
Pyle—A Manual of Personal Hygiene,
Saunders' Medical Hand-Atlases,
Saunders' Pocket Medical Formulary,
Saunders' Question-Compends,
Stewart and Lawrence—Essentials of Medical Electricity,
Thornton—Dose-Book and Manual of Prescription-Writing,
Van Valzah and Nisbet—Diseases of the Stomach,

NOTHNAGEL'S ENCYCLOPEDIA
OF
SPECIAL PATHOLOGY AND THERAPEUTICS

IT is universally acknowledged that the Germans lead the world in Internal Medicine; and of all the German works on this subject, Nothnagel's "Encyclopedia of Special Pathology and Therapeutics" is conceded by scholars to be without question the **best System of Medicine in existence.** So necessary is this book in the study of Internal Medicine that it comes largely to this country in the original German. In view of these facts, Messrs. W. B. Saunders & Company have arranged with the publishers to issue at once **an authorized edition** of this great encyclopedia of medicine in English.

For the present a set of some ten or twelve volumes, representing the most practical part of this encyclopedia, and selected by a competent editor with especial thought of the **needs of the practical physician,** will be published. These volumes will contain the real essence of the entire work, and the purchaser will therefore obtain at less than half the cost the cream of the original. Later the special and more strictly scientific volumes will be offered from time to time.

The work will be translated by men possessing thorough knowledge of both English and German, and **each volume** will be **edited by a prominent specialist** on the subject to which it is devoted. It will thus be brought thoroughly up to date, and the American edition will be more than a mere translation of the German; for, in addition to the matter contained in the original, it will represent the **very latest views of the leading American specialists** in the various departments of Internal Medicine. The whole System will be under the editorial supervision of a **clinician of recognized authority,** who will select the subjects for the American edition, and will choose the editors of the different volumes.

Unlike most encyclopedias, the publication of this work **will not be extended over a number of years,** but five or six volumes will be issued during the coming year, and the remainder of the series at the same rate. Moreover, each volume will be revised to the date of its publication by the American editor. This will obviate the objection that has heretofore existed to systems published in a number of volumes, since the subscriber will receive the completed work while the earlier volumes are still fresh.

The usual method of publishers, when issuing a work of this kind, has been to compel physicians to take the entire System. This seems to us in many cases to be undesirable. [illegible] this encyclopedia, physicians will be given [illegible] System at one time; but any single volume [illegible] obtained by those who do not [illegible] not so profitable to the pub- [illegible] **offers to the purchaser many advantages** which will be appreciated by those [illegible] at one time.

[illegible] will, without question, [illegible] **the greatest System of Medicine ever produced,** and the publishers feel con- [illegible] profession.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

L46 Eichhorst, Herman
E34e Practice of medi
1901 7300

v.2 NAME DAT

L46 Eichhorst, Hermann L.
E34e Practice of medicine
1901 7300

v.2 NAME	DATE DUE